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PRESSURE CHANGES IN THE MIDDLE EAR AFTER ALTERING THE COMPOSITION OF CONTAINED GAS

G. MELVILL JONES

Ex otorhinolaryngologica Fennica

ERRATA

In Vol 53 6 of *Acta Oto Laryngologica* page 575 the symbols in the legends to Figs 2 and 3 have been confused and you are herewith asked kindly to substitute the following

FIG 2 Labyrinthine function acute (uncompensated) monolateral destruction  = no activity

 = facilitated spontaneous activity

FIG 3 Labyrinthine function compensated monolateral destruction hot water in right ear

 = spontaneous activity  = increased and facilitated activity

Acta Oto Laryngologica

The origin of these changes is easy to understand when pressure changes occur in the environment but similar effects may be encountered up to 36 hours after flying when on the ground and in the absence of significant changes in atmospheric pressure. As in flight failure to re-establish pressure equilibrium can lead to pathological changes recognised in the clinical condition termed delayed barotrauma (Dickson *et al* 1947). The present paper describes experiments designed to investigate the mechanism of these delayed pressure changes.

METHODS

Middle ear pressure changes were measured in five ears (three subjects) after simulated flights in a decompression chamber during which approximately known gas mixtures were introduced into the middle ear space and associated air cells by

breathing known gas mixtures during recompression or "descent". During ascent middle ear gas is expelled through the Eustachian tube and during descent this is replaced through the same channel by the prevailing nasopharyngeal gas mixture, the composition of which is largely determined by that of the inspired gas. Approximately known amounts of the desired gas were introduced by adopting a standard simulated flight which required either two descents from a pressure one quarter that at ground level or four descents from one half ground level pressure, the latter procedure being necessary for maintenance of consciousness when breathing gas having a low oxygen percentage. In this way approximately 15 l/ths of the original middle ear content was replaced with new gas. The gas mixtures breathed were pure oxygen and 55%, 21% and 10% oxygen in nitrogen. These were delivered through a conventional pressure demand regulator mask system of the kind currently employed in the Royal Air Force.

Immediately after final 'descent' the subject closed his Eustachian tubes, maintaining closure for 1 to 2 hours by avoidance of talking and swallowing, the latter being facilitated by removal of saliva with a dental sucker. After 10 minutes, a delay introduced for reasons discussed later, middle ear pressure changes relative to atmosphere were measured at 10 minute intervals throughout the next hour or more, when a steady rate of fall in pressure was usually observed (Fig. 3, continuous line). The subject then returned to his normal daily occupation, re-establishing pressure equilibrium by opening the Eustachian tubes as desired, in order to avoid development of large pressure differentials across the ear drum. Roughly six hours later, the precise time being chosen by the subject, he ceased clearing his ears and returned for a second similar series of measurements with the Eustachian tubes closed. In each complete experiment this sequence of events was repeated (Fig. 3, discontinuous lines) until the rate of pressure change during a one hour series of measurements approximated to 2.5 cm/h. this being the mean of steady state values obtained from the five ears examined.

Apparatus

The apparatus employed for measurement of middle ear pressure changes was similar to that described by van Dishoeck (1941) and called by him the pneumophone. It comprised an hermetically sealed source of sound connected with the ear through a tube capable of hermetic seal in the external auditory meatus. A variable volume capsule was included for altering the pressure in the closed system, this pressure being measured with a water manometer. When a sound of constant intensity and frequency was presented to the ear in this way, it is found to have maximum loudness when the pressure in the closed system approximates to that in the closed middle ear space (Weyer, Bray & Lawrence 1952; Weyer, Lawrence & Smith 1948). The pressure corresponding to maximum loudness is easily determined and with practice a surprisingly accurate assessment of a chosen end point can be obtained. The experimental results illustrated in Fig. 1 in which each point is the mean of 10 values gave $S.D.s$ of 1.5, 0.18, 1.22, 0.58 and 1.0.5 cm H₂O at trans tympanic pressure differences of approximately 0, 10, 20, 30 and 40 cm H₂O respectively.

EXPERIMENTAL BASIS OF METHOD

The relation between change in middle ear pressure and maximum loudness pressure in the pneumophone was examined by the following

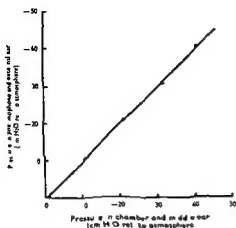


FIG 1

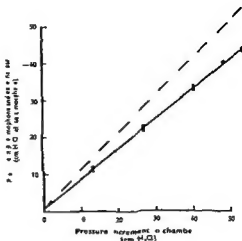


FIG 2

FIG 1 Change of maximum loudness pressure in pneumophone and external ear relative to ground level atmospheric pressure related to change of pressure in decompression chamber and middle ear Eustachian tube remaining open. Each point is the mean of 10 readings.

FIG 2 Change of maximum loudness pressure in pneumophone and external ear relative to pressure in the decompression chamber Eustachian tube closed (continuous line). Each point is the mean of 5 readings. The intermittent line gives the relation to be expected if the middle ear were a simple container having constant volume and temperature. Intersection of the ordinate above the origin probably reflects the fact that maximum loudness is usually obtained at a small pressure differential across the ear drum.

experiment. A subject was enclosed in a chamber, one external ear being connected through the chamber wall with an external pneumophone operated by an external observer who obtained maximum loudness readings on instruction from the subject. The pressure in the chamber was then progressively reduced, the subject keeping the Eustachian tube open so that his middle ear pressure would exactly follow that in the chamber, maximum loudness pressures in the pneumophone being recorded at intervals of 10 cm H₂O. Free communication between middle ear and nasopharynx was ensured by the subject maintaining the characteristically loud sound of his own voice when the Eustachian tube is open. The results of this experiment are shown in Fig 1, in which the change in maximum loudness pressure in the pneumophone and therefore external ear, is plotted against change of pressure in the chamber, and therefore the middle ear. Each point is the mean of ten readings. A straight line was obtained, the slope of which does not differ from unity at the 5% level of significance, with the conclusion that in this experiment middle ear pressure change was fairly accurately reflected in the pneumophone. Presumably maximum loudness occurs at a given balance of forces upon and therefore position of, the ear drum.

In this experiment there was no difference in pressure between the middle ear and its surroundings, whereas in practice the apparatus was mainly

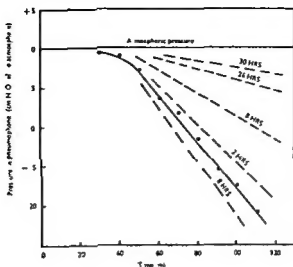


Fig. 3. Observed pressure changes during the first two hours after a flight breathing pure O_2 (continuous line). Each point is the mean of 5 readings. Subsequent rates of pressure drop are shown in the intermittent lines. Results obtained from one ear.

employed for following a relative middle ear pressure drop. A second experiment more closely simulating the main experiments was therefore performed in which after initial equilibration with his local atmosphere the subject maintained closure of Eustachian tube while the pressure in the chamber was progressively increased. For convenience the pneumophone was retained in the chamber, maximum loudness readings being taken at intervals of roughly 10 cm H_2O over a total range of 50 cm H_2O . The results are shown as the continuous line in Fig. 2 in which observed change in pneumophone pressure is plotted against increment of pressure in the chamber. Each point is the mean of five readings. A somewhat surprising feature here was that the individual regression lines from each subject were indistinguishable from one another at the 5% level of significance.

If the middle ear behaved as a simple rigid container having constant temperature and volume the absolute pressure exerted by enclosed gas would be expected to remain constant and on the basis of the previous experiment so also would that in the pneumophone at maximum loudness. The pneumophone being contained in the chamber this would then be reflected as a fall in pressure equal in magnitude but of opposite sign to the pressure change in the chamber. A line having slope unity would then be obtained (intermittent line 1 in 2). In practice a straight line having slope 0.80 emerged.

In this experiment the quantity of gas contained in the closed middle ear space must have remained substantially constant since time would not permit significant gaseous exchange. The temperature also presumably remained constant at body temperature. It transpires therefore that there are additional factors, discussed later, which in these circumstances apparently cause the

apparatus systematically to under read. The important outcome is that over the range of experiment the degree of under reading and hence the reading itself was always proportional to the total change in local atmospheric pressure. In this experiment the cause of relative pressure change was change in local atmospheric pressure the middle ear gas content remaining constant. But if the cause of the relative pressure change were gaseous exchange across the enclosing surface rather than change of atmospheric pressure presumably the outcome would be the same. In this case the observed pressure change would be proportional to the net gaseous exchange incurred and hence it is concluded that the apparatus affords a reasonably reliable means of following the phenomenon.

RESULTS

The pressure changes observed in one ear during the first two hours after a standard simulated flight breathing pure oxygen are shown in the continuous line in Fig. 3. After a short delay discussed below there was a steady fall in pressure presumably due to gas exchange across the surface lining the middle ear space the rate of fall being given by the slope of the curve. In

TABLE 1 Observed rates of middle ear pressure fall with Eustachian tube closed, after flights breathing known gas mixtures (time 0 at 30 min after completion of final descent)

Subject	100% O ₂ breathed						50% O ₂ breathed						21% O ₂ breathed						10% O ₂ breathed					
	Time (h & min)			Rates of pressure fall (cm H ₂ O/h)			Time (h & min)			Rates of pressure fall (cm H ₂ O/h)			Time (h & min)			Rates of pressure fall (cm H ₂ O/h)			Time (h & min)			Rates of pressure fall (cm H ₂ O/h)		
		L ear	R ear		L ear	R ear		L ear	R ear		L ear	R ear		L ear	R ear		L ear	R ear		L ear	R ear		L ear	R ear
J G	0	11.7	6.1	0	14.8	6.3	0	1.5	1.7	0	7.8	12.1	0	1.5	1.7	0	7.8	12.1	0	7.8	12.1	0	7.8	12.1
	5.30	18.8	18.4	1.10	21.0	16.7	4.45	5	8.5	6.0	1.0	1.6	11.45	23.3	16.0	12.55	7.3	12.0	10.30	4.0	0.9	—	—	—
	24.20	9.4	11.7	21.55	5.0	5.9	—	—	—	—	—	—	29.15	4.0	3.3	29.10	0.8	0.2	—	—	—	—	—	—
	35.35	2.9	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
D W R	0	17.0	12.5	0	15.5	14.0	0	15.3	11.7	0	5.0	4.1	1.0	19.2	20.0	1.0	20.4	21.9	4.0	6.0	8.0	4.0	0	0
	12.0	23.3	2.3	12.0	1.8	8.4	1.05	4.0	5.2	—	—	—	21.0	15.4	1.9	21.0	2.7	3.5	—	—	—	—	—	—
	30.0	1.4	2.8	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
C M J	0	18.3	—	0	18.6	—	0	15.6	—	0	5.1	—	7.15	20.5	—	5.30	1.4	—	3.55	8.8	—	6.25	0.7	—
	12.35	14.4	—	11.15	8.0	—	1.55	0.8	—	—	—	—	18.35	8.7	—	1.25	2.5	—	—	—	—	—	—	—
	21.35	4.0	—	—	—	—	—	—	—	—	—	—	29.50	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

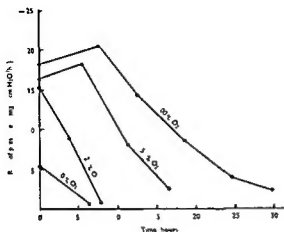


FIG. 4

FIG. 4. Time course of observed rates of pressure change after flights breathing different gas mixtures. Results from same ear as Fig. 3.

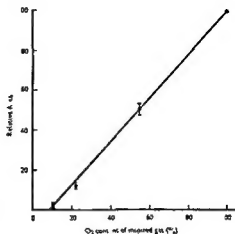


FIG. 5

FIG. 5. Relation between areas under curves similar to those in Fig. 4 and the O_2 content of gas breathed during the preceding flight, obtained from 5 ears in all experiments. Each point is the mean of 5 values; the standard error of which is bracketed. The areas are expressed as percentages of those obtained after breathing pure O_2 in each case (Relative Areas).

the same figure the slopes of similar lines obtained at 8, 12, 18, 24 and 30 hours are indicated by the discontinuous lines. After an initial delay of roughly 12 hours, also to be discussed below, the rate of pressure drop depicted by the slopes of these lines progressively declined until after 30 hours near equilibrium was obtained. Table 1 gives the rates of change obtained from the slopes of lines such as these at specified times after flights breathing specified gas mixtures for all ears examined.

From the data in Table 1 curves were drawn plotting observed rates of change against time elapsed and in this way a family of curves showing the time course of the rate of pressure change after flights breathing different gases was obtained for each ear. One such family of curves obtained from the same ear as the results shown in Fig. 3 is shown in Fig. 4. The striking feature here is the close relation seen between the areas under these curves and the corresponding percentage oxygen breathed during descent in the preceding flight. This is important because oxygen percentage here reflects the middle ear oxygen content immediately after flight whilst the areas being the integrals of observed rates of pressure change with respect to time may be thought of as the sum of all the small pressure changes which would have been observed between moments of opening the Eustachian tube had it been possible to do so continuously. In view of the results obtained in Fig. 2 these areas are therefore probably approximately proportional to the net absorption of gas across the surface lining the middle ear.

space from beginning to end of each experiment. The relation between percentage oxygen breathed during descent and corresponding areas is shown more clearly in Fig 5 in which the areas are expressed as a percentage of the pure oxygen area in each family of curves and hence are referred to as Relative Areas. Each point is the mean of five values the standard error of which is bracketed. Inspection of the graph shows that a linear relation was obtained which in turn suggests a direct relation between middle ear oxygen content immediately after flight and subsequent net absorption of gas. From this it seems likely that oxygen absorption was the main cause of the observed pressure changes. Presumably oxygen is absorbed until near attainment of equilibrium with the surrounding tissues.

A rough assessment of a normal near equilibrium value can incidentally be arrived at by extrapolation of the line in Fig 5 to its intersect with the abscissa since this point suggests the percentage oxygen which on the basis of these experiments would have to be inhaled for steady state conditions to obtain immediately after flight. Intersection occurs at approximately the 8% level and when dilution of inspired with expired gas is taken into account this gives a partial pressure of about 33 mm Hg oxygen actually introduced into the middle ear for steady state conditions. However it must be emphasised that in view both of the indirect method of approach and the limited data available this cannot be considered a very reliable estimate. Attempts were made to check this value by collecting a sample of normal middle ear gas both by a direct approach through the ear drum and via the Eustachian tube. Unfortunately owing mainly to difficulty in obtaining an effective seal round the sampling tube it was found impractical to obtain a reliable sample.

DISCUSSION

The experimental results show that after flight there is a tendency for the middle ear pressure to fall below that of the atmosphere and they suggest that absorption of oxygen is the main cause of this effect. Before drawing conclusions however the influence of four possible contributory factors require to be examined namely diffusion of gases other than oxygen, temperature change in the enclosed gas, volume change of the enclosed space and application of force upon the ear drum through the medium of the ossicular chain.

Gas Diffusion

The gases to be considered in addition to oxygen are water vapour, carbon dioxide and nitrogen. Changes in water vapour content are considered unlikely to have influenced the results significantly since saturation must be rapidly achieved, probably being complete after the new gas

already partially saturated in the tracheopharynx has traversed the Eustachian tube. After introduction of new gas having a lower partial pressure of carbon dioxide than that in the surrounding tissues, relatively rapid diffusion of this gas would be expected to occur from tissues into the space with consequent rise in pressure. In practice the new gas introduced cannot have been CO_2 free on account of mixture with expired gas in the tracheopharynx. Indeed owing to the relatively constant partial pressure of alveolar CO_2 at all altitudes some concentration of this gas is to be expected in the middle ear during descent. An experiment in which gas was sampled from a region close to the pharyngeal orifice of the Eustachian tube during the standard flight indicated a final partial pressure in the middle ear of 40 mm Hg. However, this is still 10 mm Hg below an equilibrium figure of approximately 50 mm Hg quoted independently by Campbell (1924) and Van Liew (1957) in their experiments with gas pockets under the skin and hence some initial diffusion into the space is likely to have taken place. This is thought to be reflected in the initial delay in fall of pressure referred to earlier and illustrated in Fig. 3; occasionally even an initial increase in pressure was observed. But since the determination of rates of fall of pressure upon which the main results are based necessarily preclude acceptance of data until a steady rate of fall was evident, it is considered that the effect of carbon dioxide upon the results can be largely neglected.

With regard to diffusion of nitrogen Van Liew (1957) gives data from which it is possible to compute mean rates of gas exchange after introduction of different gas mixtures under the skin. After introducing pure oxygen and after an initial delay of 40 minutes to allow carbon dioxide equilibration the N_2 excretion rate was approximately one tenth that of oxygen absorption. After introduction of 44% and 79% nitrogen in oxygen, nitrogen excretion was too slow to manifest itself over the course of two four hour experiments. These findings therefore suggest that although possibly responsible for reducing the slopes of initial lines obtained after experiments with high oxygen concentrations (Fig. 1) the contribution due to nitrogen diffusion would be too small to affect the results significantly. However there is an additional feature which may have some bearing on this conclusion, namely a tendency to plateau formation in those curves showing the time course of rates of pressure change (Fig. 4 and Table 1) associated with high oxygen content. That is to say, although the partial pressure of oxygen must have been falling due to its absorption and ultimate replacement with air through the Eustachian tube, its rate of absorption tended to remain steady where plateau formation occurred. A simple explanation would be limitation of the rate of oxygen absorption imposed by the blood flow through the local vascular bed and the oxygen dissociation curve of its contained haemoglobin. But in this case the relative significance of nitrogen excretion would probably be increased which in turn would then contribute to plateau formation and might even in part account for the reduced initial rates of pressure drop frequently observed.

Temperature Change

Theoretical considerations based on conduction of heat alone suggest that the time to attain a temperature within 1°C of the surrounding walls for a gas having initial temperature 15°C would be less than one minute and hence the influence of this factor upon results is also considered negligible

Volume Change and Force Acting upon the Drum

On the other hand the possibility of results being affected by changes in volume of the space and the application of force upon the eardrum through the ossicular chain cannot be neglected. Any distension of blood vessels or soft tissues within the bony walls of the middle ear space due to local reduction of pressure would diminish the gas containing volume and hence also the observed fall of pressure. Moreover the pressure difference across the footplate of the stapes caused by local fall of pressure in the middle ear would establish a static force presumably transmitted through the ossicular chain and acting upon the eardrum in a sense opposing the pressure gradient. This mechanism would also tend to reduce the observed fall in pressure and hence the amount by which the experimental observations are influenced must be the algebraic sum of the two effects. The results in Fig. 2 suggest however that this amount is over the range of the experiment proportional to the pressure change which would be observed in a similar container having constant volume and no ossicular chain. Consequently although the combined effect of these two factors is considerable this conclusion indicates that the final results as expressed in Fig. 5 are unlikely to have been significantly influenced since here they are expressed in a comparative form as a percentage of the values obtained with pure oxygen for each ear.

CONCLUSIONS

In view of the absence of significant influence of the factors considered above upon the results as they are presented it is reasonable to conclude that the strong correlation between middle ear oxygen content and total after flight pressure change reflected in Fig. 5 is good evidence that the observed changes are mainly due to absorption of oxygen. The higher the oxygen content of gas inhaled during flight the higher is the final oxygen content to be expected in the middle ear after flight and hence also the potential suction due to its absorption.

As already mentioned intersection with the ordinate at approximately the 8% oxygen level in Fig. 1 suggests a steady state is normally achieved when the partial pressure of oxygen has fallen to about 55 mm Hg in the middle ear. It is noteworthy that this figure is somewhat higher than equilibrium values observed in subcutaneous and intra peritoneal gas pockets (Rahn & Cranfield 1933; Campbell 1924). But in acute stenosis of the Eustachian

tube a pressure drop develops spontaneously (van Dishoeck 1944) and thus it seems that in the normal state when there is no stenosis of the tube true equilibrium conditions do not obtain. Presumably the standing difference between the sum of pressures exerted by dissolved gases and local atmospheric pressure leads to continuous slow absorption of all gases which in turn demands introduction of air through the Eustachian tube. But air contains oxygen at approximately 150 mm Hg and consequently a higher steady state partial pressure of oxygen is to be expected in the middle ear than in a closed gas pocket under the skin.

Practical Implications

There are two practical implications which may be mentioned. First if the same findings apply to a blocked sinus then in treatment by washout it might be logical to replace washout fluid with nitrogen for then oxygen would be expected to diffuse from surrounding tissues into the space thereby raising the internal pressure and thus facilitating drainage and maintenance of patency of the osseus rather than the reverse which would be expected from the introduction of air. Secondly from the standpoint of the aviator it would be an advantage to maintain the partial pressure of oxygen inspired during flight at the lowest value compatible with operational efficiency. For then the liability to post flight pathology due to repeated development of suction and maintained high partial pressures of oxygen in the middle ear would be reduced to a minimum.

ZUSAMMENFASSUNG

In fünf Mittelohren wurden Druckveränderungen nach Einfüllung von verschiedenen Gasgemischen von ungefähr 100 bis 21 und 10 Prozent Sauerstoff mit Stickstoff untersucht. Die Druckveränderungen wurden mittels eines Apparates ähnlich dem von Dishoeck sehen Pneumophon beobachtet. Bei geschlossener Eustachischen Membran fiel der Druck mit einem Gradienten von 1 bis zu 230 mm WS/h ab. Mit wiederholter Gaszufuhr durch die Eustachische Röhre verringerte sich dieser Gradient über eine Zeitsdauer von 1 bis zu 30 Stunden. Eine Gesamtwertung der Ergebnisse zeigt eine lineare Beziehung zwischen dem Gesamt Druckabfall (d.h. Zeitintegral über die beobachteten Einzelgradienten) und dem prozentualen Sauerstoffgehalt des eingeführten Gemisches. Hieraus lässt sich schließen dass die Sauerstoffabsorption die Hauptursache des beobachteten Druckabfalls war. Die Ergebnisse weisen ferner auf einen Druck von ungefähr 55 mm QS als normalen Partialdruck des Sauerstoffes im Ruhezustand im Mittelohr hin.

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PRIMARY MALIGNANT GROWTHS OF THE TRACHEA

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Primary cancer of the trachea is a rare condition. Between 1943 and 1959 19 cases were seen at the Christie Hospital & Holt Radium Institute 16 of these having histological proof. The main clinical findings are described and treatment is discussed. Results of treatment confirm the lethal nature of the disease—only one case of this series has lived longer than 5 years.

The occurrence within a few weeks of each other of two cases of primary malignant disease of the trachea stimulated our interest in this rare condition. A search of the records in the Christie Hospital & Holt Radium Institute between 1943-59 revealed a total of 19 cases, 16 of which were histologically proved. As the only other reports of a series of cases in the literature have all come from the Mayo Clinic—Jung (1930), Olsen (1939) and Finney, Moersch & McDonald (1943)—it was felt worth while to present the main clinical findings of these 16 cases with a brief discussion of the modes of treatment and the results thereof.

Culp in 1938 published a review of the literature up to 1936 and collected 147 cases of primary carcinoma of the trachea, 82 of which had been proved histologically. To emphasise the rarity of the condition he pointed out that only two cases had been found in 47,600 autopsies at North American hospitals. Illman & Whitaker (1947) reviewed the literature between 1935 and 1945 and added 38 cases of histologically proven primary malignant neoplasms of the trachea to the 82 already noted by Culp, giving a total of 120 cases.

The rarity of primary neoplasms in the trachea contrasts with the incidence of carcinoma in the larynx and bronchus. Fruhling & Spehler (1951) reported three primary tumours of the trachea in 60,000 biopsies examined at Strasbourg between 1921-50 while during the same period there were 740 cases of carcinoma of the larynx and 243 cases of carcinoma of the bronchus. In the period 1943-59 there were registered at the Christie Hospital & Holt Radium Institute 313 cases of bronchial carcinoma and 1415 cases of laryngeal carcinoma. While the incidence of carcinoma of the bronchus has increased considerably, there is no suggestion of a similar trend in tracheal cancer.

It is sometimes difficult to be certain whether a tumour is primary in the trachea or has invaded from a neighbouring organ. The two most likely

TABLE I

Primary site	Total no	Tracheal invasion	
		Definite	Possible
Oesophagus	175	8	3
Thyroid	141	6	11

sources from which such invasion might occur are the thyroid gland and the oesophagus. Any such origin has been excluded as far as possible in all the cases quoted here as primary neoplasms of the trachea and we have examined the case notes of a consecutive series of cases of malignant growths of the thyroid and oesophagus to see how commonly invasion of the trachea occurred. The results are given in Table I.

Thus it seems that confusion as to the primary site of a tracheal tumour is most likely to arise in the upper third where it is in relation to the thyroid gland.

Age incidence

This is a disease of middle age and our figures correspond closely with those published by others. The average age was 51 years, only one patient being over 60 years old and the youngest being 37 years of age.

Sex incidence

Ten out of our sixteen patients were males. Culp quotes the incidence as 63% males.

Site

Culp found that the lower third was the commonest site of origin but in the 14 cases in our series where it was possible accurately to determine this the upper third was the site in six cases, the middle third in five and the lower third in three.

Pathology

Filman & Whitaker analysed 120 histologically proven primary malignant neoplasms of the trachea recorded up to 1945 and found the following distribution:

Adenocarcinoma	56
Squamous cell carcinoma	50
Basal cell carcinoma	14

In our 16 cases the histology was as follows:

Squamous cell carcinoma	11
Cylindroma	2
Basal cell carcinoma	1
Adenocarcinoma	1

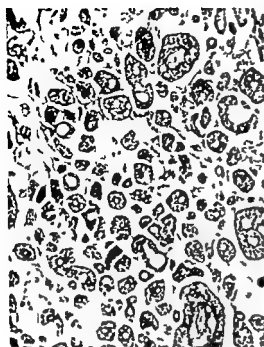


FIG. 1 Cylindroma of the trachea ($\times 11 \times 1$) (Magnification $\times 13$)



FIG. 2 Basal cell carcinoma of the trachea ($\times 11 \times 1$) (Magnification $\times 100$)

Other types of malignant growths are reported rarely, some of them being merely differences in nomenclature.

FIGS 1 and 2 are microphotographs illustrating a cylindroma and a basal cell carcinoma respectively.

Spread

Metastases are common in this condition and Culp found they had occurred in 62 out of 91 reported cases. Ten of our 16 cases had metastases, the distribution being as follows:

Supraclavicular lymph nodes	5
Deep cervical lymph nodes	2
Lungs	2
Cerebral	1

Spread to mediastinal nodes probably occurs frequently, but in this series proof of such spread was lacking; in only two cases did X-ray examination suggest that this had occurred. A post mortem was performed on one case where widespread dissemination was found in the liver, lungs, suprarenals and lymph nodes.

Symptomatology

Dyspnoea and cough are the dominant symptoms (Tinnex *et al.* 1943). In our 16 cases 9 patients complained of dyspnoea and in five instances this

was associated with stridor. Cough was present in 8 cases and 7 of these had haemoptysis. Other symptoms were hoarseness (5 cases) and dysphagia (5 cases) but this latter symptom usually occurred only in the later stages of the disease. Jackson (1936) stated that an asthmatoïd wheeze was an important symptom and Tinney *et al* found this in 36% of their cases. In our series it occurred in three patients and in two cases this led to an initial mistaken diagnosis of asthma.

The presenting symptoms in our series in order of frequency were

(1) Dyspnoea and/or stridor in 7 cases. In three of these there was an associated asthmatoïd wheeze. In one instance the patient also presented with a lump in the neck.

(2) Hoarseness in 4 cases, one of which had an inspiratory stridor.

(3) Cough in 4 cases, three of whom also had haemoptysis.

(4) One case presented as acute respiratory obstruction which necessitated an immediate tracheotomy.

The duration of these symptoms before attendance at hospital varied from one month to four years but was usually less than six months.

Signs

Apart from the stridor shown by five cases there were few physical signs. A mass could be felt in the suprasternal notch in two cases and supraclavicular nodes were present in five instances. Examination with a laryngeal mirror revealed a recurrent nerve paralysis in one case, the tumour may be seen between the cords if it is in the upper third and this was so in three instances in our series. Auscultation of the chest may reveal a wheeze over the trachea and emphysema or collapse in one or other lung if a ball valve effect has occurred (Jackson) but this has not been noted in any of our cases.

Diagnosis

Tumours in the cervical trachea can usually be diagnosed without much difficulty by the combined use of the laryngeal mirror and soft tissue plain X rays of the neck. The report below is a good example of such a case.

E J L. Male aged 54 years. Occupation commercial traveller.

First seen 3.9.59. Complaints 8 months increasing difficulty in breathing, 9 months fatigue and some loss of weight. No dysphagia. His own practitioner had treated him for asthma during the past year.

On examination: an anxious man with marked inspiratory and expiratory stridor at rest. On viewing the larynx with a mirror a mass could be seen below the cords projecting from the posterior wall of the trachea, the main mass being slightly to the right of the midline. Both vocal cords were mobile.

Further investigations: blood count normal, chest X ray normal. A soft tissue lateral view of the neck showed a rounded soft tissue projection from the posterior wall of the trachea $1\frac{1}{2}$ below the cords (see Fig. 3). A barium swallow showed a slight indentation of the anterior wall of the oesophagus opposite the tracheal mass.



Fig. 3



Fig. 4

but no definite evidence of invasion. Tomography confirmed these findings, the tracheal tumour being well shown on several of the views.

10.9.59 Direct laryngoscopy: vocal cords appeared normal and both were fully mobile. There was an irregular mass arising from the posterior tracheal wall and extending downwards for about 2 cm. The mass extended laterally, more on the right side than on the left. Biopsies were taken and these were reported as showing squamous cell carcinoma.

It was decided that he should be treated by radiotherapy, and in view of his already narrowed airway, a low tracheotomy was performed on *14.9.59* between the fifth and sixth cartilaginous rings, at which level no evidence of the tumour could be seen through the tracheostomy opening. A plastic tracheotomy tube was fitted and his growth treated with the 4 million volt linear accelerator, the total tumour dose being 5750 rads over 21 days, the treatment starting on *2.10.59*.

There was a brisk reaction to the irradiation and he developed some dysphagia. When seen one month after the cessation of treatment, he looked well. There was considerable oedema of the arytenoids, but no slough could be seen in the trachea and a soft tissue view of the neck showed some reduction in the size of the soft tissue shadow in the trachea. The cords were moving satisfactorily and he had a reasonable airway.

20.2.60 Bronchoscopy and pharyngoscopy revealed no evidence of any remaining neoplasm and he was debulked. There was some initial intermittent stridor after this but it improved rapidly. He was last seen on *23.6.60* when his condition remained satisfactory and X-rays showed a good tracheal airway with no obvious recurrence (see Fig. 4).

The diagnosis of lesions in the thoracic trachea can be difficult. Plain X-rays often fail to reveal any abnormality, though views taken after the

instillation of iodised oil and tomography may be helpful. For instance in four cases the initial X-ray examination was negative and in two of these this led to an unfortunate delay in diagnosis.

Endoscopy is the usual means of diagnosis in these cases and should always be performed if there is any suspicion of such a lesion. Biopsies can be taken and a good idea of the nature of the tumour and its site can be obtained.

Treatment

This has usually been by radiotherapy, surgical excision, diathermy, coagulation or a combination of these methods. Tinney *et al* recommend that growths in the upper third be treated by tracheal fissure and electrocoagulation while those in the lower two thirds should be destroyed via the endoscope with electrocoagulation; and the gland fields treated by deep X-ray therapy. Barrett (1958) states 'the risks of recurrence are so great that the surgical operations which have been attempted must be regarded as palliative procedures'.

In our 16 cases 4 were considered unsuitable for any treatment due to the advanced stage of the disease. One case was palliated successfully for two years by repeated endoscopic coring out of the tumour. The other eleven were all treated by radiotherapy.

Irradiation of the trachea to radical dosage presents problems which are worth while discussing briefly.

(1) The trachea is, except in the neck, a relatively deep seated organ and before the introduction of supervoltage equipment delivery of a tumour lethal dose was not easy; also the position of the shoulders relative to the thorax makes satisfactory treatment planning difficult.

(2) The oesophagus lies in close proximity and care must be taken to avoid overdosage lest a most unpleasant, painful and protracted oesophagitis result. In addition it is important to avoid over irradiation of the lung parenchyma which was a real problem when many cross firing X-ray beams generated at 250 kV were required. The dangers of the resulting lung fibrosis are well known.

(3) In most cases the ideal volume of tissue to be raised to tumour dose is a cylinder whose centre is the estimated centre of the tumour and which covers the tumour with at least a 2 cm margin all round. This treated volume is difficult to attain elegantly because of the backward obliquity of the organ in relation to the anterior chest wall.

It is our practice now to treat these tumours over three weeks on the 4 MV linear accelerator. This machine has two important advantages as compared with conventional equipment: (i) the depth dose of the X-rays produced is so much greater—fewer fields are therefore required, less lung is irradiated and the integral dose is much reduced; and (ii) the ability to regularise the geometry of this part of the body by the use of wedge filters facilitates treatment planning and the achievement of a cylinder of high dose around the trachea.

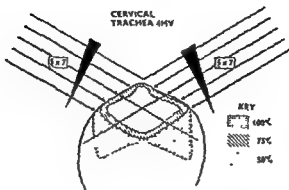


FIG 5

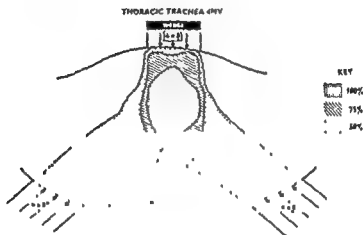


FIG 6

Field sizes are of the order of 8×11 cm and the tumour dose 5500–5750 rads. Reactions have not been severe and no serious complications have been noted.

Figs 5 and 6 illustrate two techniques, one for tumours at the upper end of the trachea and the other for middle and lower third lesions, which we have found to overcome substantially the problems mentioned.

Results

The results of treatment by any means are poor, the usual expectation of life from the time of the first diagnosis being 6–12 months. Tinney *et al* reported, however, that of the 8 cases of cylindroma treated at the Mayo Clinic, one had lived 10 years, and the other 7 cases were alive and well when last heard from, three of them having then survived 5 years. Of the 11 cases of squamous cell carcinoma, only one was alive and well nine years after treatment by diathermy and deep X ray therapy.

Houel, Lebon & Collège (1958) searched the literature in France, Great Britain and U.S.A. for the preceding 10 years and found 17 reported cases of

primary tracheal tumours treated by surgical excision six of which were alive and free of disease, the longest survival being 3 years

Only one of our 16 cases lived beyond 5 years finally dying of the disease seven years after the first diagnosis. This patient was treated for the first year by repeated endoscopic diathermy destruction of the tumour, and then had radiotherapy, 5000 rads being given in one month. The histology in this case was said to be a basal cell carcinoma possibly arising from the duct of a mucous gland.

As previously stated, 4 cases were not treated because of the advanced stage of the disease. Of the remaining 11 cases 7 are dead the average length of survival after treatment being eleven months.

Four cases treated recently are still alive, three of them free from disease twenty six, seven and three months respectively after treatment, while the fourth is alive seven months after treatment but with evidence of disease. The case which has so far survived for 26 months is a cylindroma of the upper end of the trachea.

Remarks

Malignant disease of the trachea is a rare and lethal condition. Most surgeons see only a small number of these cases in their career and as the diagnosis, especially of lesions in the thoracic trachea, is difficult, mistakes occur. The diagnosis should be considered in any middle aged patient complaining of increasing dyspnoea, cough and haemoptysis. Negative findings on plain X-ray can be misleading and endoscopy should be performed in any case where doubt exists.

The small number of cases treated by any individual makes the assessment of results difficult, but from a study of the literature and the cases at the Christie Hospital & Holt Radium Institute it would appear that cases of cylindroma of the trachea are the only ones with a reasonable prospect of cure. Other types of malignant growth are usually rapidly fatal whatever form of treatment is used, though individual successes using different forms of treatment, have been reported.

ZUSAMMENFASSUNG

Primärer Tumor der Trachea kommt selten vor zwischen 1943 und 1950 hatte man im Christie Hospital & Holt Radium Institute nur 16 Fälle, davon 16 mit histologischen Beweisen. Die klinischen Befunde werden beschrieben und die Behandlung besprochen. Die Behandlungsergebnisse erharteten die Letalität der Erkrankung — nur ein Fall dieser Serie verblieb länger als fünf Jahre am Leben.

ACKNOWLEDGEMENTS

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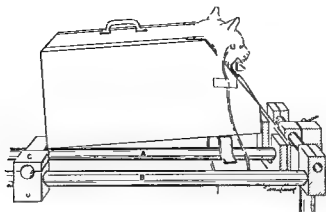


FIG 1 Side view of the animal box. The box containing the animal is fixed on the tilting table. Any position in the vertical plane can be achieved by tilting the frame A at the axis C. In this study (for calorization) frame A was tilted 45 deg above the horizontal plane, thereby bringing the horizontal canals of the cat into optimal position. Frame B permits rotation of the tilting frame.

space must be reproducible so that comparable data, with caloric tests, can be obtained. The second aim was to study the nystagmic reaction in normal cats under different conditions of caloric stimulation.

METHOD

Fixation of Body and Head

The present study was carried out on 52 adult, healthy cats. Fixation of the body was achieved by wrapping it tightly with a towel. The body was then enclosed in an animal box which could be fixed on a tilting device as illustrated in Fig 1. The tilting table insures an accurate reproduction of any position from one test to another.

Fixation of the head was done in the following manner. Under ether anesthesia transverse holes were drilled with a 1 mm dental burr, in the base of each upper canine. After recovery from anesthesia the body was placed in the animal box. A thin steel bar (1 mm in diameter) was inserted through the holes in the teeth and fixed to the box as illustrated in Fig 2. The introduction of the steel bar and its fixation to the box could be done rapidly and simply. The cats accepted it without any sign of discomfort.

Stimulation

The animals were brought into an optimal position for caloric stimulation, by tilting the front of the box 15° above the horizontal plane.

A constant temperature circulator was used for irrigation (Fig 3). This circulator pumped a continuous stream of water through the output rubber tube and back again into the waterbath. With this arrangement the water and tubes were maintained at equal temperature, thereby minimizing the loss of heat during irrigation. The nozzle, consisting of 3 mm diameter polyethylene tubing, could be introduced easily into the external canal. Water temperatures of 20°, 28°, 30°, and 48°C were

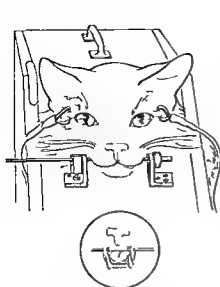


FIG 2

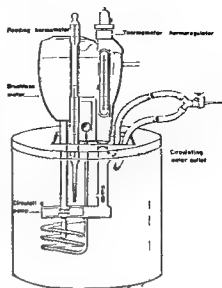


FIG 3

FIG 2 Immobilization of cat's head. The diagram enclosed in the circle illustrates the steel bar inserted through the holes of upper canines. The front view of the cat shows fixation of the steel bar to the box by means of brackets on both sides of the head. The hook electrodes for recording corneo retinal potentials are inserted in the canthi of the orbits.

FIG 3 Diagram of a constant temperature circulator which is commercially available (*Science* 130: 1130, 1959). When the nozzle is clamped the water continues circulating throughout the tubing system.

employed. During irrigation care was taken to prevent the outflow from the external ear from dripping into the box; otherwise, artifacts on the electrical recording could occur.

In all tests the duration of irrigation was 40 sec. Within this interval the outflow of the pump through the nozzle located in the external canal was 75 cc of water.

Recording of Nystagmus

Recordings were made with a modified EEG two-channel pen writer. One channel, whose time constant was ≈ 0 sec, recorded the conventional nystagmic movements. The other channel had a time constant of 0.01 sec (Henriksson, 1955), and recorded the velocity of the slow component. This tracing is called the derived nystagmus. The corneo retinal potentials were picked up from the external canthi of the eyes by means of hooks implanted in the skin (see Figs. 1 and 2).

The speed of the recording paper could be set at either 1.5, 3, 6, 15, 30 or 60 mm/sec. For recording the caloric reaction, the paper was usually set at 1.5 mm/sec. A total caloric reaction could then be represented on a 200 mm length of paper. For the recording of optokinetic eye movements the paper was set at 11 mm/sec.

Calibration

When the cat was exposed to a pure optokinetic stimulus (rotation of constant angular velocity) the animal often seemed to ignore it, which resulted in irregular

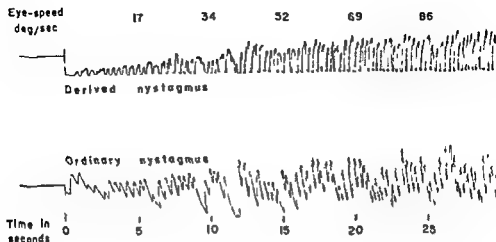


FIG. 4. Calibration. The 'ordinary nystagmus' represents the reaction to rotation at 3.4 deg/sec^2 in full light. The velocity of the slow component is recorded as the 'derived nystagmus'; these tracings are calibrated in degrees per second according to the method described in the text. The speed of the paper was set at 6 mm/sec .

eye-movements, but if vestibular as well as optokinetic stimuli were applied by means of a constant angular acceleration, then a clear cut nystagmus was obtained. Under this condition, the eye velocity of the slow component was assumed to be equal to the angular velocity of the rotating table (Henriksson, 1956). This equality seems to hold at least for small accelerations ($2\text{--}4 \text{ deg/sec}^2$) which last for a short time (less than 20 sec). The angular velocity of the rotating table can be calculated in degrees per second at any point on the tracing, within this interval, simply by multiplying acceleration by time. From this the corresponding velocity of the slow component in the derived tracings is defined, resulting in a graded curve in degrees per second. Such a curve can be used as a reference for quantitative evaluation of other derived curves, for example, that obtained by a caloric test.

Fig. 4 shows both ordinary and derived nystagmus during an acceleration of 3.4 deg/sec^2 in full light. The tracing of the ordinary nystagmus records both slow and fast components, and that of the derived nystagmus records the slope of the tracings of the slow component. In other words the latter represents the velocity of the eye movement during the slow component. For convenience, in the course of this paper, the velocity of the slow component will be called "eye-speed".

Evaluation of Nystagmus Tracings

The caloric reaction is generally evaluated by its duration. Some authors measure its intensity in terms of frequency times amplitude (Ohm, 1939), others by total number of jerks (Hood & Pfaltz, 1954), and still others by the maximum eye speed (Henriksson, 1956). In the present paper the caloric reactions were evaluated by both the nystagmus duration and maximum eye speed. The latter was obtained by comparing it with the optokinetic curve. Owing to variations in the derived nystagmus with position of the electrodes, it is necessary to obtain a reference curve for each application of the electrodes.



FIG. 2. Photomicrograph of one canine showing the hole drilled at its base. The animal was sacrificed 12 days after the dental operation. The diameter of the hole is 1 mm leaving a supporting structure for the steel bar of about 1 mm in each side. H.E. $\times 9$.

RESULTS

A. Efficiency of the Fixing Device

A point of much concern during the experiments was whether the dental holes produced inflammatory reactions. Any discomfort resulting from this procedure would not only influence the results from caloric stimulation but it could affect the general health of the animals. At the beginning of this investigation the animals rested for several days after the dental operation. Later it was found that this precaution was unnecessary for the cat could be fixed immediately after the operation without resistance or signs of discomfort. No interference with feeding was ever observed. As a matter of fact the animals gained weight during the course of the experiment. The photomicrograph of Fig. 2 shows a longitudinal section of a canine 12 days after the hole had been drilled. Whereas bone dust and a microscopic hemorrhage was found in the dental pulp there was no evidence of caries, pulpitis or periapical periodontitis.

Fixation of the cat as described in the method provided most satisfactory tracings of corneo-retinal potentials following caloric stimulation. Those artifacts induced by small movements during irrigation however could not be avoided (see Figs. 7 and 9). Likewise after irrigation some animals also presented body movements contaminating the tracings with artifacts.

Fixation and testing were successfully conducted in adult cats of various sizes. The procedure failed in kittens and adult cats with defective canines. Throughout the entire fixation procedure there were only 4 of 22 animals in

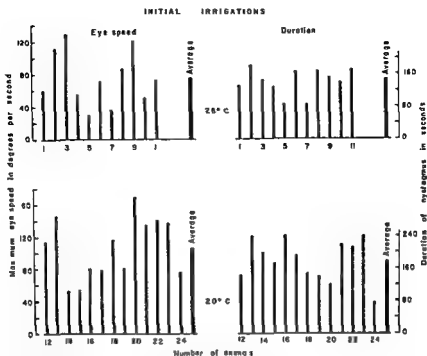


FIG 6 Nystagmic reaction to first caloric test. The graph presents the data of the nystagmic reaction of eleven cats (upper row) irrigated with water at 28°C and that of 13 cats (lower row) irrigated with water at 20°C. Notice that the latter are numbered from 12 to 24 and that the scale of eye speed column is different from that of duration.

which the teeth were broken. These failures were due to inexperience of the experimenters in either passing the steel bar through the canines or fixing of the head to the animal box.

The mounting of the animal box to the tilting table (see Fig 1) enabled the investigators to place the cats in an optimum position for caloric stimulation. Of equal importance, this position could be reproduced accurately from one test to another.

B Nystagmic Reaction to Caloric Tests

Pilot studies revealed that the first caloric test did not always produce a nystagmic response. Moreover, the nystagmic reaction, when present, varied frequently from one test to another. These irregularities were attributed to technical difficulties in irrigating the external canal. More specifically, the tympanic membrane of the cat could not be readily visualized because of the S shaped external ear and the presence of obstructing cartilages of the auricle. In view of these anatomical arrangements, one could not be certain that the irrigation was being directly applied to the eardrum. This particular problem was solved by designing a plastic operation which exposed the tympanic membrane. Four cats were tested under these conditions, but again the nystagmic responses could not always be evoked. If present, they showed

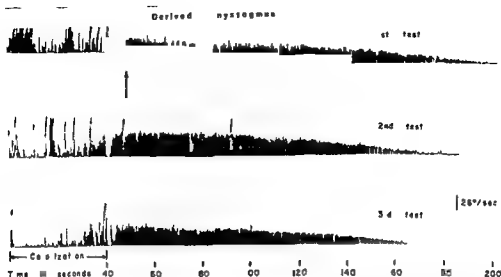


FIG. 7. Nystagmographic records of three consecutive irrigations with water at 20°C in the same ear. During the calorization small movements of body and/or head appear as large deflections in the tracings; thereafter minimum artifacts are seen. The upward displacement of the strip ahead of the arrow is an artifact produced by faulty centering of the pen. Calibration: 26 deg per sec. Speed of paper: 1.5 mm/sec.

marked irregularities. This finding then ruled out the possibility that difference in placement of the irrigating nozzle was responsible for the inconsistencies in nystagmic reactions. Two other possibilities remained: inappropriate temperature of the irrigating water and the effect of repeated caloric stimulation.

1. Effect of water temperature

In this series 24 cats not previously exposed to caloric stimulation were used. Irrigation with water at either 8°C below or above rectal temperature of the cat, i.e. 30° or 46°C, failed to produce recognizable nystagmus. Decreasing the water temperature to 28°C always elicited a nystagmic reaction. There was, however, considerable variation among animals in both maximum eye speed and duration of nystagmus, which is illustrated in the upper row of Fig. 6.

In a series of 13 cats, nystagmic reactions were clearly present following irrigation with water at 20°C. The results presented in the lower row of Fig. 6 also varied among animals; however, the maximum eye speed and duration of nystagmus were in most instances greater than those recorded in the previous series.

2. Effect of repetitive caloric tests

In this series the effect of caloric tests repeated at five minute intervals was studied under four conditions:

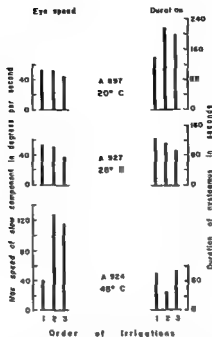


FIG. 8 Nystagmic reaction to repeated caloric test as function of temperature. For each temperature the test was conducted three times in the same ear. In the central column is the animal number and the temperature of the water used for irrigation. The graph indicates that the nystagmic reaction for individual animals can be reproduced although irregularities in both maximum eye speed and duration may occur.

(a) *Repetitive tests in the same ear with constant temperature* Caloric stimulation was repeated three times, with different animals being used to study the effect of water temperature at 20°, 28° and 48°C. Fig. 7 shows the nystagmographic records obtained in one animal whose ear was irrigated with water at 20°C. This cat, as well as those tested with water at 28° or 48°C, (Fig. 8) exhibited adequate and reproducible responses. Evaluation of the results in terms of both maximum eye speed and duration of nystagmus, demonstrated either a slight response decline or irregularities in the nystagmic reaction.

(b) *Repetitive tests in alternating ears with the same temperature* Three cats were given six tests each, with the irrigations alternating between the right and left ear. In one animal, the caloric test was conducted with water at 48°C while in the other two, water at 20°C was used. Fig. 9 presents the records of a case irrigated with water at 20°C. The figure shows that the speed of the slow component diminished with repetitive tests, while the duration of nystagmus was not apparently affected. These changes in the caloric responses were not as evident in the animal tested with water at 48°C (Fig. 10).

(c) *Repetitive tests in the same ear with hot and cold water* Two animals were used and each received six tests: three cold (28°C) alternating with three hot (48°C). The results presented in Fig. 11, revealed that the maximum

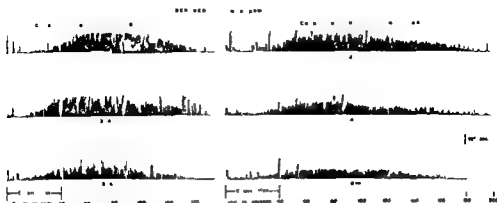


FIG. 9 Nystagmographic records of six consecutive irrigations with water at 20°C alternating between right and left ears. The order of the irrigation is indicated at the bottom of each tracing. The records show a moderate response decline in the speed of the slow component when the caloric test was repeated. Calibration 52 deg per sec. Paper fed at 15 mm/sec.

eye speed varied considerably from one test to another while the duration of the nystagmus remained approximately the same.

(d) *Repetitive tests in alternate ear with hot and cold water* The caloric test in this series was carried out in the same manner described by Fitzgerald & Hallpike (1942). Each of three cats was exposed to cold and hot water irrigations in the following order: right 28°, left 28°, right 48°, and left 48°C.

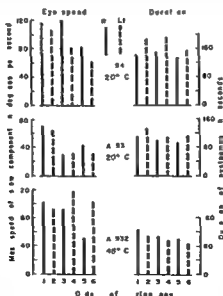


FIG. 10 Effect of repeated caloric tests alternating between right and left ears. Each animal was irrigated six times with water at one temperature. The solid lines indicate that the calorization was done on the right ear while calorization on the left is indicated by broken lines. A response decline in maximum eye speed is evident in those animals irrigated with water at 20°C.

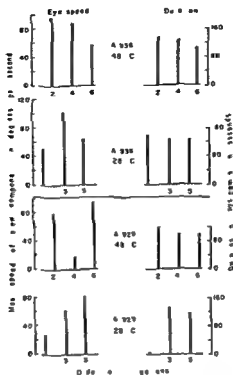


FIG. 11 Effect of alternating irrigations cold and hot in the same ear. The order of the irrigation is indicated by numerals on the abscissa. The data shows considerable variability in maximum eye-speed. The duration of nystagmus in cat A 929 to the first irrigation with water at 28°C is not recorded because its measurement was uncertain.

This sequence was repeated three times. A representative case is illustrated in Fig. 12. The upper part of the figure shows that the maximum eye speed decreased progressively with repetitive tests. The lower part representing the classical form of graphing the caloric test of Fitzgerald & Hallpike (1942) also shows a consistent response decline in the duration of nystagmus.

DISCUSSION

These experiments demonstrate that nystagmographic recording of caloric stimulation in the cat can be obtained when both body and head are properly immobilized. The method of fixation is simple; the animal shows little or no signs of distress, and movement artifacts in the nystagmographic tracings are reduced to a minimum. This technique can also be used advantageously for recording both spontaneous and positional nystagmus, optokinetic nystagmus, and that provoked by a rotatory test.

It is generally accepted that the technique of Fitzgerald & Hallpike (1942) for a caloric test in man provides an accurate measure of the receptor sensitivity. This method can now be applied to the cat for similar purposes, provided the temperature of the irrigating water is at least 10°C above or 10°C

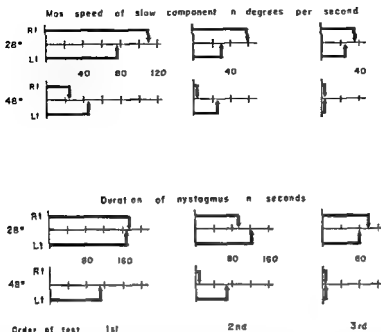


FIG 12 Repeated Fitzgerald-Hallpike's caloric test in the cat. Plottings of either the maximum eye speed or duration of nystagmus show a pronounced response decline with repetitive testing. The duration of the nystagmus in the first test with water at 48°C in the right ear is not recorded because of uncertainty in its determination.

below the rectal temperature of the animal (38°C). The evidence suggests strongly that in the cat, any caloric test should not be repeated unless necessary, since consecutive irrigations produce an unmistakable decline in responsiveness. This response decline is more apparent for the maximum eye speed.

The test results with repetitive cold and hot water irrigations in the same ear, indicate that alternating ampullofugal and ampullopetal flow causes considerable variation in the maximum eye speed, however, the duration of the nystagmus is little affected.

The results of repeated irrigations with either cold or hot water in one or both ears show a decremental effect upon the nystagmic reaction, particularly with respect to the maximum eye speed. The response decline becomes increasingly apparent as the sequence of irrigations is repeated. This phenomenon of response decline to caloric stimulation is presently under investigation.

ZUSAMMENFASSUNG

In einfacher Plan für das Fixieren des Körpers und des Kopfes in einer Katze wurde vorgenommen um die Nystagmus Reaktion an den Caloric Reizungen zu untersuchen. Die Nystagmographen in den gewöhnlichen und auch abstammenden Nystagmusen wurden erhalten relativ frei von Artifact, verursacht durch Bewegung.

Um in einer Katze eine Caloric Reaktion zu verursachen war es notwendig die Temperatur des Spulwassers mindest 10 C unter oder über der Rektal Temperatur (38°C) zu bringen

Wiederholte Caloric Reizungen mit kaltem und auch heissem Wasser wurden in verschiedenen Umständen vorgeführt und die Ergebnisse beschrieben. Diese Ergebnisse weisen den zweckmassigsten Weg um zuverlässige Resultate mit der Caloric Examination in einer Katze zu bekommen.

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VERTIGO AND NYSTAGMUS RESPONSES TO CALORIC STIMULI REPEATED AT SHORT INTERVALS

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Repeated monaural caloric stimulations with water of constant temperature at intervals of 10 minutes brought about a progressive decline of the vertigo and nystagmus responses as measured by the mean values of the latency, duration and maximum intensity of the vertigo, the duration of the nystagmus and the total number of nystagmic beats.

The calorization series were paired so that they differed in regard to (1) only the labyrinth (left or right), (2) only the temperature of the water (warm or cold), or (3) both the labyrinth and the temperature of the water. The maximum intensity of the vertigo in the second of a paired series appeared to be independent of that in the first series, regardless of the type of pairing. The decline in the vertigo response thus appeared to show a directional specificity linked to the direction of the cupular deflexion in the individual lateral semicircular canal.

INTRODUCTION

The present investigation was undertaken to study some patterns of the vertigo and nystagmus responses to repeated monolabyrinthine caloric stimulations.

The *vertigo response* to repeated caloric stimulations has been the object of only a few systematic studies. Jongkees (1933) stated that the caloric induced vertigo has no clinical value. Hamersma (1957) and Preber (1958) on the other hand found that young subjects usually have the ability to analyse the turning sensations with fairly great accuracy.

The *nystagmus response* has been somewhat more extensively studied. Dunlap (1935) reported that the nystagmus responses of two rabbits to repeated monaural stimulations with ice water showed a progressive decline and that the response to stimulation of one labyrinth was diminished if preceded by a series of stimulations to the opposite labyrinth. Spiegel & Aronson (1933) reported that in cats kept under superficial anaesthesia continuous caloric stimulation can produce nystagmus for several hours. This observation was confirmed by L'ecroite de No (1934). Loch & Haines (1946) found in five subjects that stimulation with small amounts of ice water at intervals of 24 hours produced no change in the duration of the nystagmus response.

Mittelman (1954) reported interindividual variations in the responses of 24 normal subjects to three to six monolabyrinthine stimulations with 10 ml of water at 17°C at intervals of 15 min. The total amplitude was influenced more often than the total number of beats while the duration showed no uniform change.

A brief mention of the responses to *repeated rotatory stimulation* would seem to be in place. Griffith (1920) found a progressive decline of the nystagmus as well as of the apparent motion in man. Mowrer (1932-1934) reported that the nystagmic responses of experimental animals showed a progressive decline provided the rotations were iso directional. Hallpike & Hood (1953) reported that repeated iso directional rotations produced a decline of the vertigo response and considered that the development of the R D phenomenon is dependent upon a directional specificity of cupular adaptation. Hood & Pfaltz (1954) however who reported a decline of the nystagmic responses of rabbits stated that the R D phenomenon was dependent upon the number of stimuli applied and not upon the interval between them. They were able to confirm the observation of Fearing & Mowrer (1934) that the R D phenomenon does not develop if the stimuli are applied during light anaesthesia. They therefore considered it likely that its mechanism depends upon some central process involving consciousness. Guedry *et al* (1957, 1958-1959) who studied the post rotatory vertigo responses reported that their experimental results showed systematic departures from the data predicted by the torsion pendulum theory as applied to the cupula endolymph system by van Egmond *et al* (1949-1952). They considered that the R D phenomenon is due to an adaptation in the vestibular system probably dependent upon a central factor.

Caloric stimulation cannot be applied with the same precision as the rotatory but has the advantage of permitting the study of the responses from each labyrinth separately. To my knowledge no one has hitherto taken advantage of this possibility in the study of the R D phenomenon.

MATERIAL AND METHODS

The experiments were performed on 25 healthy subjects between the ages of 19 and 31 years. There were 10 men and 15 women (13 medical students, 10 physiotherapy pupils and 2 college students).

All subjects fulfilled the following requirements: (1) no past history of vertigo, auditory impairment or tinnitus; (2) normal appearance of the auditory canals and tympanic membranes on inspection; (3) no evidence of spontaneous positional or abnormal gaze nystagmus on examination with the eyes closed and open; (4) normal or almost normal routine calorigram according to the criteria advanced by Stahle (1956-1958); (5) distinct vertigo response to at least half of the routine caloric stimulations; (6) ability to analyse and describe the turning sensations accurately.

The subjects were instructed not to take any drugs (especially barbiturates and antihistamine agents) or alcohol, not to attend tired or otherwise indisposed and not to discuss their experiences with each other.

The apparatus was essentially the same as that described by Aschan (1955) and Aschan, Bergstedt & Stahle (1956). Water at constant temperatures of 30° and 14°C

was supplied from two separate water baths each furnished with an electrical heater, a contact thermometer and a built in stirrer. Determinations of the temperatures of the water with an electrical thermometer showed that during the passage through the rubber tube from the water bath to the ear there was a heat loss of 0.8° in the 44°C water but no measurable loss in the 30°C water.

The electronystagmograms were recorded from silver cup electrodes filled with electrode jelly and fixed at the outer canthus of each eye with adhesive tape. The electrodes were connected to a battery driven Grass P4 pre amplifier and then to an ELEMA two channel Mingograph 24 (power amplifier with liquid jet writing recorder), the amplifiers push pull coupled. The time constant was 1.6 sec and the paper speed 1 cm/sec.

All tests were performed with the subject in the supine position the head ante flexed 30° from the horizontal and the eyes closed. Approximately 150 ml of water was injected into the external auditory canal over a period of 30 sec, an interval of 10 min being allowed to elapse between tests.

In the routine caloric tests four stimuli were applied right ear with cold water, left ear with cold, right ear with warm left ear with warm.

In the experiments a series of four to six stimuli was applied to the same ear with water of the same temperature. As a rule two series of stimuli were applied during an experimental session and paired in one of the following ways (a) first series to one ear and the second to the opposite ear, both with water of the same temperature (Type 1) (b) first series with cold water and the second with warm water, or vice versa, both to the same ear (Type 2) (c) first series with cold water to one ear and the second with warm water to the other ear or vice versa (Type 3). In the total series Types 2 and 3 were commenced equally often with warm water as with cold. An interval of 10 min was allowed to elapse between the two series.

The vertigo response was assessed with regard to latency, duration and maximum intensity. The latency was measured from the commencement of the irrigation to the onset of the vertigo and the duration from the onset of the vertigo to its termination. The maximum intensity was assessed with the guidance of the following descriptions of the vertigo response given to the subjects and are based on my own experiences during experiments performed on myself.

- + + Very intense turning sensation accompanied by a sensation of being thrown off the couch Unpleasant
- () = Intense turning sensation possibly slightly unpleasant
Moderate turning sensation distinct in direction and plane Not unpleasant
- () Mild turning sensation possibly not fully distinct in direction and plane
= No turning sensation but distinct sensation of rocking or swaying
- () = Vague sensation of rocking or swaying
- 0 = No sensation of movement of the body in relation to the surroundings

This is a seven point evaluation scale which in the records was recorded as + + + - - - () - 5 and so on. The term speed was used synonymous with intensity.

The nystagmus response was assessed with regard to latency, duration, number of beats and rhythm (degree of dysrhythmia). The latency was measured from the com

mencement of the irrigation to the first detectable nystagmus beat and the duration from the first to the last detectable beats. The number of beats denotes the total number of distinct nystagmus beats. Now, not all nystagmus beats are equally distinct; the rhythm of nystagmus is often somewhat irregular. Thus nystagmus dysrhythmia denotes that periods of distinct nystagmus beats alternate with periods of less distinct or undiscernable beats. The degree of dysrhythmia has been roughly evaluated as follows:

- Grade 3 - Alternation between short periods of distinct nystagmus and considerably longer nystagmus free intervals
- Grade 2 - Alternation between about equally long periods of distinct nystagmus and nystagmus free intervals
- Grade 1 - Alternation between long periods of distinct nystagmus and considerably shorter nystagmus free intervals
- Grade 0 - Regular or almost regular nystagmus

RESULTS

A. Vertigo

In the routine caloric tests all the subjects stated that they experienced vertigo with all four irrigations. Since these tests involved the training of inexperienced subjects to describe vertigo with special regard to the maximum intensity they will not be gone into more fully. In the following I shall confine myself almost entirely to the results of the experiments but since certain qualities of the calorically induced vertigo are not dependent on any one form of experiment I shall mention something about them here.

The direction of the turning sensation was with few exceptions the same as that of the quick phase of the nystagmus. This observation is in agreement with the findings of earlier workers (Wodak (1953), Hamersma (1957), Preber (1958)).

The plane of the turning sensation Like Hamersma (1957) I found that the majority of the subjects stated that rotation was either round the long axis of the body or round a sagittal axis through the umbilicus. Sometimes the turning sensation was of a complex nature with changes in direction and plane during a response.

The intensity of the turning sensation was in most cases characteristic with a rapid initial increase to a climax and a relatively slow decline. This is in good agreement with Stahl's (1956) observation regarding the nystagmus response.

The vertigo responses within a series of caloric stimuli

A total of 78 series of four to six irrigations were performed. 14 subjects received four series, 2 subjects received three series, 7 subjects received two series and 2 subjects received one series. The uneven distribution of series among the subjects is due to technical difficulties and humane considera-

TABLE 1 *Vertigo in response to successive monaural irrigations with water of constant temperature*

Arithmetical means (M) and standard deviations (σ) for the latency, duration (in seconds) and maximum intensity in series of four successive irrigations

Irrigation no	Latency		Duration		Maximum intensity	
	$M \pm \sigma / \sqrt{n}$	σ	$M \pm \sigma / \sqrt{n}$	σ	$M \pm \sigma / \sqrt{n}$	σ
1	30.62 \pm 1.37	10.14	65.50 \pm 2.86	24.92	3.57 \pm 0.10	0.91
II	33.73 \pm 1.68	12.47	49.34 \pm 2.72	23.69	2.78 \pm 0.13	1.17
3	41.29 \pm 1.34	9.92	42.43 \pm 2.83	24.69	2.29 \pm 0.16	1.37
4	44.09 \pm 1.48	11.00	35.76 \pm 3.57	31.16	1.76 \pm 0.17	1.44

tions. In all series the vertigo response was assessed with regard to duration and maximum intensity, in 55 series the latency was measurable.

The mean values for the vertigo responses to the first four irrigations of a series showed a progressive increase of the latency and a progressive decrease of the duration and maximum intensity (Table 1).

In 43 series there was an increase¹ of the latency, and in 31 series a decrease¹ of the duration. In 64 series a decline of the maximum intensity occurred. Only in two series performed on the same subject (K.E.), was there an increase of the maximum intensity. In two series the intensity of the vertigo remained at a constant level and in ten series it fluctuated. In 24 instances the vertigo disappeared during the course of a series at the second irrigation in six series, at the third irrigation in nine series and at the fourth irrigation in nine series.

The method employed for assessing the maximum intensity of the vertigo was found to be of practical value in that the symbols used for the different degrees of intensity never needed to be altered afterwards in order to do justice to the subject's conception of the relative intensity of the response from irrigation to irrigation.

The vertigo responses in paired series

Seventy-four of the caloric series were paired in the manner described earlier. Type 1 in 11 instances, Type 2 in 12 instances and Type 3 in 14 instances. The comparison between the vertigo responses in the first and second series of a paired series was limited to the maximum intensity, since the R.D. phenomenon is most easily recognized in this factor.

On commencement of the second of a paired series the R.D. in the first series was, as a rule, succeeded by an increase of the response (Table 2). This break in the general tendency of the R.D. of vertigo may be regarded

¹ Increase¹ denotes that no succeeding value was less than the preceding value and that the last value in a series was greater than the first. Decrease¹ denotes that no succeeding value was greater than the preceding value and that the last value in a series was less than the first.

TABLE 2 *Differences between the maximum intensity of the vertigo response to the last caloric stimulus of the first series and the first stimulus of the second series in a total of 37 paired series*

1 grade difference—the subject experienced a clear difference 2 grades—the subject experienced a very distinct difference

Change of max intensity of vertigo	Paired series			Total n=37
	Type 1 n=11	Type 2 n=12	Type 3 n=14	
Increase 2 grades	9	9	8	26
Increase 1 grade	2	2	3	7
No difference	0	1	2	3
Decrease 1 grade	0	0	1	1

as a sign of independence between the two series paired. Furthermore it may be accepted that no difference between the last response in the first series and the first response in the second series does not indicate either independence or dependence between the paired series and that an initial decrease in the second series is a sign of dependence. If we accept this reasoning we find that in paired series Type 1 eleven second series are independent of the first and none is dependent in Type 2 eleven are independent and none dependent and in Type 3 eleven are independent and one appears to be dependent. Just this last instance however occurred in the subject in whom a series of stimuli produced a progressive increase of the vertigo. In analogy with the foregoing reasoning we thus find that also in this case the second series may be regarded as independent of the first. Table 2 can then be presented as follows—

	Type 1	Type 2	Type 3
Independent	11	11	12
Dependent	0	0	0

For Types 1 and 2 (11 cases each) the probability that no cases of the dependent category occur by chance is 1% or less and for Type 3 (12 cases) 0.1% or less.

The response patterns in the first and second series of a paired series were usually essentially the same and often congruous. The mean values for the first four responses in all first series was 2.64 and in all second series 2.48. For obvious reasons no statistical analysis of these values has been done but the difference between the mean values may be termed slight or inconclusive.

In eight experiments the effects of 6 and 10 minutes interstimulus intervals on the response pattern were compared and found to show no difference. From the physiological point of view however the 10 minute interval would seem to be less

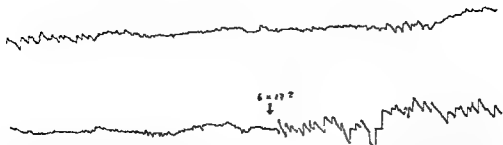


FIG. 1 Sudden verbal presentation of an arithmetical problem promptly elicits a regular nystagmus from a state of severe dysrhythmia. The lower record is a direct continuation of the upper record.

open to criticism. This is supported by the observations of Cawthorne & Cobb (1954), who measured the temperature changes within the lateral semicircular canal in man in response to irrigation of the external ear with water at approximately 20.5° and 41.5°C over a period of forty seconds and found that it took about 10 min for the temperature to return to the initial level.

B Nystagmus

In routine caloric tests the criterion of a normal nystagmus response has been applied not only to the duration and number of beats but also to the rhythm. A nystagmus response with moderate or severe dysrhythmia has been regarded as suspicious of abnormality. This is because a number of otologists consider that severe nystagmic dysrhythmia suggests dysfunction of the vestibular system (cf Stahle 1958). The present experiments however have provided ample evidence that severe nystagmic dysrhythmia cannot always be relied upon to reflect abnormal labyrinthine function. For example an arithmetic problem presented during a period of severe dysrhythmia promptly elicited regular nystagmus (Fig. 1 cf Mahoney *et al.*, 1957). This clearly shows that mental factors may play a role in the calorically induced nystagmus.

My personal experience of the caloric tests and questioning of the subjects have shown how amazingly little one is conscious of the eye movements at least so long as attention is concentrated on the vertigo. Consciousness of the nystagmus appears to diminish during the course of a series of stimulations. Instructions to let the eyes move freely or to keep them still had little or no effect on the nystagmus.

Nystagmus responses within a series of caloric stimuli

In 41 of the 76 series it was possible to measure accurately the latency, duration and number of beats in each of the four tests comprising a series. In the majority of the remaining series there was such marked dysrhythmia that an assessment of these three factors may be regarded as uncertain and arbitrary and has therefore not been attempted.

TABLE 3 *Nystagmus in response to successive monaural irrigations with water of constant temperature in 41 series (n) of four successive irrigations*

Irrigation no	Latency		Duration		Number of beats	
	$M \pm \sigma / \sqrt{n}$	σ	$M \pm \sigma / \sqrt{n}$	σ	$M \pm \sigma / \sqrt{n}$	σ
1	23.49 \pm 1.12	7.16	154.3 \pm 5.6	35.84	198.5 \pm 9.7	61.69
II	28.12 \pm 1.01	6.49	140.2 \pm 3.3	21.11	180.0 \pm 7.9	50.47
3	27.02 \pm 1.19	7.62	140.0 \pm 4.4	27.85	178.3 \pm 7.8	49.89
4	26.90 \pm 1.10	7.05	136.1 \pm 4.0	25.69	168.3 \pm 8.2	52.44

As shown in Table 3 there was a progressive decline in the mean duration and number of beats during the course of a series of stimulations while there was no regular change in the latency.

In the total series there was an average nystagmic dysrhythmia of Grades 0 to 1 in 44 series and of Grades 2 to III in 34 series. In 76 series no definite increase or decrease of the dysrhythmia could be observed while in 20 series there appeared to be a definite increase and in 2 series a definite decrease during the course of a series.

The nystagmic responses in paired series

Since a progressive decline of the nystagmus responses occurred in only a minority of the series the material is too small for any conclusions with regard to a possible directional specificity in the response pattern. A rough comparison of the average grades of nystagmic dysrhythmia shows, however, that in six paired series the dysrhythmia was definitely greater in the second series than in the first and in two paired series it was greater in the first than in the second. In the remaining 29 paired series there was no definite difference.

C. Relationship between Vertigo and Nystagmus

As mentioned earlier the direction of the turning sensation generally corresponded to that of the quick phase of the nystagmus and the intensity of the turning sensation often paralleled the intensity in the nystagmus. Furthermore the decline of the vertigo during the course of a series on the whole paralleled a concomitant decline of the nystagmus.

The vertigo, however, had a longer mean latency and a shorter mean duration than the nystagmus (cf. Tables 1 and 3). The inter-individual differences were large. In a few cases there was practically no nystagmus (extremely severe dysrhythmia) in the presence of distinct vertigo. The reverse was of course more common.

Thus the relationship between vertigo and nystagmus is of a complex nature. This is further illustrated by the following case (Fig. 2).

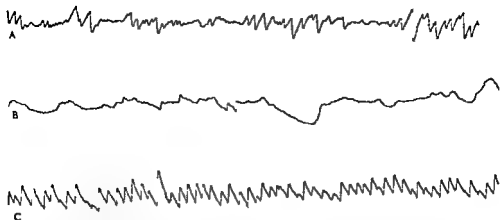


FIG 2 (Case R B) Nystagmic dysrhythmia of (A) Grade 2 in response to caloric stimulation of the right labyrinth with water at a temperature of 30°C (B) Grade 3 in response to the third stimulation of the left labyrinth with water at 30°C only a few beats of a right beating nystagmus are seen (C) regular nystagmus in response to the succeeding caloric stimulus

R B, a 26 year old medical student, was subjected to Type 1 paired series of caloric stimulations. The first series, which consisted of six irrigations of the right ear with water at 30°C, evoked a moderate nystagmic dysrhythmia which declined during the course of the series concomitant with a decline of the maximum intensity of vertigo from Grade 4 to Grade 0. After an interval of 10 min the second series of six irrigations with water at 30°C was applied to the left ear. The first three irrigations evoked severe nystagmic dysrhythmia concomitantly with a decline of the maximum intensity of vertigo from Grade 4 to 3 to 2. The fourth irrigation produced an increase of the vertigo to Grade 4 and simultaneously the nystagmus became completely regular and remained so until the end of the series while the vertigo decreased from Grade 4 to 3 to 2.

DISCUSSION

During the course of a series of caloric stimulations a decline was observed considerably more often in the maximum intensity of the vertigo than in either the duration of the vertigo and nystagmus or in the number of nystagmic beats. The present investigation has therefore been devoted mainly to a study of the maximum intensity of the vertigo. An equally extensive study of the other qualities of the vertigo and nystagmus responses would require a much larger experimental material. Such a study is in the course of preparation. The present material is not very suitable for detailed statistical analyses.

The most interesting question that arises is whether the mechanism of the R D phenomenon depends on a central and/or peripheral process. A clinical experimental investigation can only provide indications and not proof that the hypotheses are correct. The results of the present investigation indicate that both the vertigo and the nystagmus responses are centrally controlled.

1. The mean values for the six systematically observed factors in the vertigo and nystagmus responses (Tables 1 and 3) show that in five factors (all

except the latency of the nystagmus) there was not only a progressive change but a steeper change initially than in the remainder of the course of a series. This phenomenon calls to mind Dodge's (1923) observation of the correspondence between the shapes of the habituation and learning curves.

2 In one of the subjects (K E) there was an increase of the intensity of the vertigo during the course of a series of calorizations and in addition, evidence that the two paired series (Type 3) were independent of each other. The response increase must be regarded as dependent on a central mechanism (sensitization) and the process in question may be assumed to be directionally specific in analogy with the diametrically opposite, considerably more common response decline (habituation) within a series of calorizations. Unfortunately the investigation in this subject had to be interrupted owing to the presence of nausea and could not be complemented with paired series Types 1 and 2.

3 An arithmetical problem given during a period of severe nystagmic dysrhythmia can evoke regular nystagmus (Fig 1 of Mahoney *et al.* 1957). This clearly shows that calorically induced nystagmus is under cerebral influence. Nystagmic dysrhythmia was interpreted by McLay *et al.* (1957) as an expression of intermittent cerebral inhibition of nystagmus. According to this hypothesis a gradual increase of the dysrhythmia could be interpreted as an expression of progressive cerebral control of nystagmus (habituation).

So far as I am aware this problem does not appear to have been studied by neurophysiological methods. An interesting anatomical aspect however is provided by the studies of Engstrom (1958) and Engstrom & Wersäll (1958) who report that there are two distinct different nerve terminals in the vestibular end organs. These endings are distinguished by differences in size, granules and contact with the sensory cells. The agranular endings are thought to represent the afferent system and the richly granulated to have an efferent, probably inhibitory function. This would provide the possibility of a central control of impulses from the sense organs (cf Granit and Hårdh (1952) regarding the muscle spindles, Granit (1955) regarding the retina, West & Hagström (1955) regarding the olfactory bulb and Hårdh *et al.* (1956) regarding the organ of Corti).

ZUSAMMENFASSUNG

Alle zehn Minuten wiederholte monoaurale kalorische Reizungen mit Wasser konstanter Temperatur bewirkten eine fortschreitende Abnahme der Vertigo- und Nystagmus-Reaktionen mit den Mittelwerten der Latenz, der Dauer und der maximalen Intensität des Schwindelgefühls, der Dauer und der Schlagzahl des Nystagmus gemessen.

Die Serien der kalorischen Reizungen wurden gepaart insofern, dass sie sich in Betreff 1) nur des Labyrinths (links oder rechts), 2) nur der Wassertemperaturen (warm oder kalt) oder 3) beides des Labyrinths und der Wassertemperatur unterschieden. Die maximale Vertigointensität in dem zweiten Serienpaar schien unbeachtet

des Paarungstyps, von der der ersten Serie unabhängig zu sein. Die Abnahme der Vertigoreaktion schien also auf eine direktionale, mit der Richtung der cupularen De- flexion des individuellen lateralen Bogenganges verbundene Spezifität hinzuweisen.

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THE USE OF POST AURICULAR FLAP IN MASTOID SURGERY

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Zollner-Wullstein's technique in the operative treatment of otitis media chronica makes it possible for the surgeon to remove all diseased tissue and in addition to reconstruct the cavum tympani in such a way as to improve hearing. This new operative technique must be regarded as satisfactory as far as the cavum tympani is concerned and it has now become standard. But the problem of drying out the mastoidal cavity and completely restoring the epithelium is still great. Statistical calculations for large numbers of operations indicate that secretion for the cavity still occurs one year after operation in 20-30 per cent of cases. Frequently also cavities which have become dry begin to secrete again after a considerable length of time the epithelium is destroyed and granulations form. Thus lengthy period of secretion is also a danger to the part of the graft covering the cavum tympani. It appears that cavities covered with free graft may secrete longer than cavities where graft was not undertaken. Several surgeons have ceased to use free graft and instead allow the mastoidal cavity to epithelialize by means of a pedicle flap obtained from the external auditory canal.

The core of the problem is to cover the cavity with a solid resistant epithelium which can withstand inevitable secondary infection. The literature of the past years shows evidence of considerable current interest in this problem.

The operative technique described here is an attempt to cover the mastoidal cavity and the cavum tympani with a pedicle flap in which the vascular supply is retained during the critical period when the graft is growing onto the bone in the mastoidal cavity. A graft with natural vascular supply will be more capable of forming a solid resistant epithelium. The operative technique is no more time consuming than the usual method of free grafting and requires no other skills on the part of the surgeon than those employed in tympanoplasty generally.

The Technique

An approximately elliptical incision is made behind the ear but the ellipse is not completed in that a pedicle of about 2-3 centimetres in width is left near the top in a posterior direction. The incision is continued downwards to a point slightly below the lobe to ensure that the flap is sufficiently long. Beginning with the anterior section of the incision the flap together with its subcutaneous fat is dissected free of the underlying tissue and folded back.



FIG 1



FIG 2

The flap is then kept covered with a compress moistened with physiological saline. The method of incision is shown in Fig 1.

The soft tissue and the periosteum are cut through adjacent to the outer ear, and an incision is made backwards on a level with the upper edge of the auditory passage so that the soft tissue and periosteum may be folded back from the processus mastoideus. A normal radical mastoidectomy and reconstruction of the cavum tympani is now undertaken according to the methods described by Zollner and Wullstein. When this part of the operation is completed, the upper sharp edges of the bone round the mastoidal cavity are rounded off. The pedicle flap is then cut into shape. All subcutaneous fat tissue is removed from the section of the flap which is to lie in the cavity, but the author uses a full skin flap. For type IV a small hole is cut in the flap corresponding to the point where it is to lie over the foramen ovale. The pedicle flap is then laid in the cavity with its raw surface towards the bone (Figs 2 and 3).

The author has always succeeded in obtaining a flap long enough to reach some distance up the anterior wall of the auditory passage without difficulty. The flap is held in place with spongostan and a tampon moistened with an antibiotic solution. As will be seen from the sketch, a section of the roof of the cavity will be left uncovered by the pedicle graft. A slit is cut corresponding to the place where the flap is to pass through the subcutaneous tissue and the periosteum, so that the flap may pass through unhindered. Part of the cavity further down is covered by the pedicle graft obtained from the external auditory canal. The periosteum and the subcutaneous tissue are sutured over the cavity in the usual way (Figs 4 and 5).

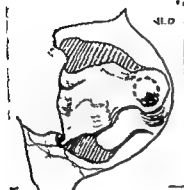


FIG 3



FIG 4



FIG 5

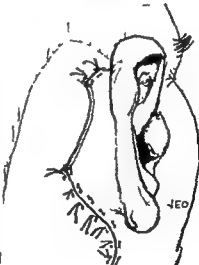


FIG 6

After the skin is freed from the subcutaneous tissue, in order that it may be stretched it is sutured no stitches are made in the pedicle, however, nor has the epithelium on the pedicle been removed so far (Fig 6)

In all operations Horner-Stoke plastic operations of the outer orifice of the external auditory canal were used. The sutures are removed 6-7 days after the operation.

Twelve days after the operation the pedicle flap is cut. The cut is made as

deep as possible in the subcutaneous tissue and the skin behind the outer ear is sewn onto the pedicle. The tampon is then removed and the following day the spongostan is also removed.

Between October 1958 and August 1959 the author performed 50 ear operations according to the technique described above. No technical difficulties arose and the operating time was no longer than that taken by the technique using free grafts.

Though the number of patients so far treated is small and it is too early to collect statistical material from comparison with tympanoplasty in which free grafts were used, the postoperative results have been very encouraging. Most of the cavities became dry and completely epithelialized within 6 months.

It would have been advantageous to have undertaken histological investigations of the flap used in this method with special reference to vascular supply, but the necessary apparatus was not at the author's disposal.

Compared with the normal technique using free graft, the operational technique described above seems to offer many advantages.

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INFLUENCE OF MATERNAL DIABETES ON THE INNER EAR OF THE FOETUS

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This is a report of histological changes found in the temporal bones of a full term infant whose mother was suffering from severe diabetes mellitus. Pronounced degenerative changes were found throughout the labyrinth, engorgement throughout the blood vessels and haemorrhages. These findings are in agreement with two cases reported by Kelemen and give reason to assume that the vascular changes so commonly present in diabetes may also involve the blood vessels of the inner ear, resulting in permanent damage of the acoustic vestibular apparatus.

For a hundred years diabetes mellitus has been known to be an aggravating factor in inflammatory diseases of the external and middle ear. The fulminant course with propagation of middle ear inflammations to the labyrinth and meninges—often tempting to excessive surgery which has proved unavailing as the patients died in sepsis or diabetic coma—was radically altered by the advent of insulin and later of sulphonamides and antibiotics.

The question whether and how diabetes mellitus affects the inner ear has also been a subject of discussion particularly towards the close of the past century. This discussion however has been purely hypothetical as practically no pathological studies were available. Kelemen (1955) found only four cases in the literature. Of these four cases three were completely predominated by severe inflammatory changes. The fourth patient described by Wittmaack (1906) was a 10 year old girl who died in diabetic coma. Wittmaack found degenerative changes of the cochlear nerve and spiral ganglion as well as granular breakdown and hyaline globules in the organ of Corti. The vestibular nerve, the maculae and cristae ampullares did not appear to be involved. Wittmaack interpreted the condition as the cause of the nerve deafness encountered in patients with severe diabetes mellitus. In Wittmaack's own case however neither otoscopy nor hearing tests had been performed but the patient's family and her own doctor reported that her hearing had been poor. Kandler (1955) however, doubts that these pathological changes are specific of diabetes mellitus interpreting the findings as artefacts. He points out that such severe changes would have involved total deafness.

After the publication of Wittmaack's case in 1906 no light was thrown on

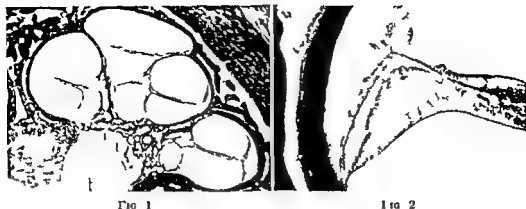


FIG 1

FIG 2

FIG 1 Cochlea with a degenerated organ of Corti and bleedings in the tympanic scala PAS haematoxylin $\times 10$

FIG 2 Detail from Fig 1 showing large bleeding in the tympanic scala $\times 90$

the relationship between diabetes mellitus and the inner ear from the pathological point of view until Kelemen in 1955 and 1960 published his findings of temporal bone changes in foetuses of mothers with severe diabetes. This is all the more remarkable as the general picture of diabetes mellitus had completely altered after the introduction of insulin. Today interest attaches mainly to the late complications of diabetes: retinopathy, nephropathy, neuropathy, coronary sclerosis and sclerosis of the peripheral vessels of the limbs. Lundbæk (1953) considers these different complications to be manifestations of a specific angiopathy characterized *inter alia* by deposition of mucopolysaccharide like substances. These changes have been demonstrated by PAS staining by the McManus method and as mentioned in an earlier publication (Jørgensen 1960) they have been demonstrated in blood vessels from synovial membranes, nerves, eyes and limbs of diabetic patients. Whether such angiopathy may occur in the inner ear is not known at present. This appears to be likely and in support of this view the present author reported on two patients (1958) suffering from long standing diabetes mellitus accompanied by severe retinopathy. The retinal bleedings were accompanied by sudden severe loss of inner ear function followed by partial restoration of hearing paralleling the ocular symptoms. In order to elucidate this problem we have collected a material of temporal bones from diabetic patients and this material is being investigated in our laboratory.

To the otologist the question regarding the influence of maternal diabetes upon the foetus is of course of great interest especially with regard to the study of the congenital disturbances of acoustic vestibular function. Kelemen (1955 and 1960) has described his findings in the ears of two foetuses. One was from a 26 year old mother with diabetes since the age of 17 years. Her pregnancy had been interrupted by hysterotomy in the sixth month because of severe progressing retinopathy. The other foetus was derived from a 24

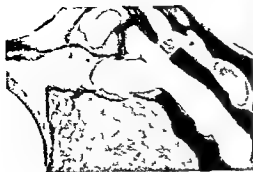


FIG 3



FIG 4

FIG 3 Bleeding in modiolus and engorged blood vessel PAS haematoxylin $\times 96$

FIG 4 Same as Fig 3 Kultschitzky staining $\times 96$

year old mother with an eight year history of diabetes whose pregnancy was interrupted in the fourth month, also by hysterotomy. In this case the indications were hyperemesis, diabetic neuropathy, anxiety neurosis, and pyelonephritis. In both cases he found widespread vascular damage in the form of haemorrhages around the small blood vessels in the vestibule as well as around the cristae ampullares in some places with disintegration of the cupulae. Furthermore, bleeding was found in the modiolus, cochlea, and between the nerve bundles in the eighth nerve. Since Kelemen's publications are the only ones of their kind on this subject I felt that it would be of interest to report a case from our laboratory, since our findings accord with his.

Case Report

A 27 year old gravida III was admitted to the Maternity Department B, University Hospital, Copenhagen because of diabetes mellitus and diabetic retinopathy. The patient had been suffering from diabetes for 22 years, from the age of five, and had been treated by insulin and diet all the time. There had been numerous hypoglycaemic episodes, but coma only once, when she had appendicitis complicated by peritonitis. During her present pregnancy her condition had been checked every ten days at a diabetic hospital. The pregnancy had been uneventful. At the time of delivery her blood pressure was normal, the urine contained sugar, but no protein. The patient was suffering from retinopathy, but there were no signs of nephropathy or neuropathy. Apart from episiotomy necessitated by a cicatricial perineum she was delivered without complications of a live boy, 3750 g and 53 cm. The placenta was delivered 5 minutes later, weight 1200 g. It was large, oedematous, loose and lobular without infarctions. The infant cried only feebly after birth, developed increasing cyanosis and had to be placed in an incubator. Despite energetic treatment with oxygen, penicillin, and Alevars the cyanosis increased and the baby died 20 hours after birth.

Autopsy was carried out at the University Institute of Pathological Anatomy, which gave the following report. Typically an infant born of a diabetic mother with



FIG 5



FIG 6

FIG 5 Ampulla of lateral semicircular canal with partially disintegrated cupula PAS haematoxylin $\times 39$

FIG 6 Collapsed utricle with disintegrated macula PAS haematoxylin $\times 39$

plump extremities, short neck, and puffy skin. No malformations. Umbilical cord normal. Site of cervical and thoracic organs, and pericardium normal. Heart of normal size and shape. Ostiae and valves normal. No congenital malformations. Pleura and trachea normal. A few petechiae superiorly on both sides. Lungs small and dark with almost total atelectasis. Sites of abdominal organs and peritoneum normal. Liver of normal size and shape, without focal changes. Pancreas and spleen normal. Adrenals, kidneys, urinary tract, external and internal genital organs normal. The infant is full term. After opening the skull we found no bleedings of the meninges and no ruptures in the tentorium or falx. Cerebrum and cerebellum grossly normal. Microscopic examination of pulmonary tissue: Atelectasis without definite focal changes.

The left temporal bone was removed, fixed in formalin and at our laboratory decalcified in Kristensen's formic acid sodium formate solution and embedded in celloidin. Serial sectioning in the horizontal plane, thickness 20μ . Every tenth section was stained with haematoxylin-eosin and selected sections were treated by Kultschitzky's myelin sheath staining and PAS staining by the McManus method in order to demonstrate changes, if any, in the vessel walls.

Observations

The middle ear showed a development normal for the infant's age with ossified auditory ossicles. It was partially air filled, since the foetal mesenchyme was present only as a broad layer on the periosteum and ligaments. In the mesenchyme dilated capillaries with ruptured walls and perivascular minor bleedings in several places.

Inner ear. The bony labyrinth was everywhere fully developed and of normal appearance. In the mesenchyme of the perilymphatic spaces there were throughout engorged blood vessels and in several places minor haemorrhages. The utricle, saccule, and semicircular canals as well as ampullae were collapsed. There were only remnants of the cupulae and maculae. In a few places erythrocytes in the lumina. The anterior labyrinth also showed pronounced engorgement of the blood vessels along the walls of the vestibular and tympanic scalae. The anlage of the cochlear canal was present in its entire length but it was greatly collapsed. The sustentacular cells in

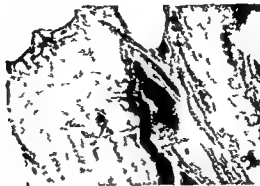


FIG. 7. Acoustic nerve and the Scarpa ganglion. Engorged blood vessel and bleedings in the nerve. Kultschitzky staining. $\times 49$.

the limbus were distinctly visible in most places but the remnants of the organ of Corti were highly disintegrated and in some places completely lost. The tectorial membrane was in most places pressed down against the underlying structure. No trace of the Reissner membrane. In the stria vascularis engorged capillaries. The neuroepithelium was disintegrated in several sites lost and replaced by connective tissue so that there was no longer a demarcation from the mesenchyme in the spiral ligament. In the PAS stained preparations the sustentacular tissue was well preserved. In the modiolus too there were large tortuous vessels in several sites showing ruptures and large perivascular bleedings which had oozed in between the ganglion cells in the Rosenthal canal. Moreover there were several extravasations around the blood vessels in the acoustic nerve. In Kultschitzky stained preparations the myelin sheaths were intact throughout and there was no reduction in the number of ganglion cells neither in the spiral ganglion nor in that of Scarpa. Nowhere thickened vessel walls or deposits of PAS positive matter.

Comments

These findings agree with those described by Kelemen viz fragility of the blood vessels with widespread haemorrhages throughout the ears. The destructions were so widespread that restitution can hardly be imagined to have been possible neither in the vestibular nor in the cochlear part of the labyrinth. Thus had the infant's life been saved the acoustic vestibular function would have been extinguished for ever. It may be asked of course whether the changes are attributable exclusively to the maternal diabetes as anoxia and stress might have given rise to a generalized bleeding tendency. It might be pointed out however that Kelemen's preparations were derived from foetuses delivered by hysterotomy in which any traumatization had been carefully avoided. And most of the changes found in the present case must also have occurred prior to the delivery since anoxia and bleeding could not have caused such widespread destructions in the 20 hours which passed from birth to death. It seems reasonable to assume that vascular changes which sooner or later affect any diabetic will also affect the foetuses of diabetic mothers.

ZUSAMMENFASSUNG

Es wird den histologischen Befund in den Felsenbeinen eines vollgeborenen Kindes einer Mutter mit einer schweren Diabetes beschrieben. Das histologische Bild zeigte schwere degenerative Änderungen überall im Labyrinth und dazu überall Stauung in den Blutgefässen sowie Hamorrhagien. Dieser Befund stimmt mit zwei früheren Fällen von Kelemen überein und lässt vermuten, dass die bei Diabetes häufig vorkommenden vascularen Änderungen auch die Gefässe des inneren Ohres angreifen können, und vielleicht Dauerschaden des akustisch-vestibulären Apparates verursachen können.

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WISSBILDUNG DES STAPFS BEI DER DYSTOSTOSIS MANDIBULO FACIALIS

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Es wird über einen Patienten mit Dystostosis mandibulo facialis berichtet. Außer den charakteristischen Veränderungen des Gesichtsschädels und der Augenlider bestand bei intaktem äußeren Ohr und Trommelfell eine Mittelohrschwerhörigkeit beiderseits. Während der Operation, die ein gutes funktionelles Resultat ergab, wurde ein deformierter Stapes gefunden, der außer durch eine Ankylose im ovalen Fenster zusätzlich durch eine knöcherne Verbindung zum Facialiskanal fixiert war.

Die Dystostosis mandibulo facialis (Dmf) auch Franceschetti Zwahlen Syndrom oder in der angelsächsischen Literatur als Treacher Collins Syndrome bezeichnet, findet in den letzten Jahren zunehmendes klinisches Interesse.

Auf die Mittelohrschwerhörigkeit als häufiges Teilsymptom dieser Entwicklungsstörung wurde kürzlich von Clerc & Deumier (1958) hingewiesen. Nach der Darstellung von Eigenbeobachtungen bei dem Syndrom von der Hoeve und der Dystostosis cranio facialis (Crouzon) besprechen die Verfasser die Möglichkeit der Stapesmißbildung oder einer Mißbildung der labyrinthischen Kapsel im Bereich des ovalen Fensters bei der Dmf. Ihre Vermutung, daß die Schwerhörigkeit bei dieser Erkrankung nicht nur durch eine Atresie des äußeren Gehörganges, sondern zusätzlich durch eine Stapesankylose bedingt sei, wurde schon bei Bregant & Naud (1949) ausgesprochen.

Es sollen die Veränderungen beschrieben werden, die wir in der Paukenhöhle eines Patienten mit einer Dmf fanden.

Anamnese

Die Eltern des Patienten leben und sind gesund, es besteht keine Blutsverwandtschaft. Eine Großtante mütterlicherseits war schwerhörig. Erbkrankheiten oder angeborene Mißbildungen kommen auch in der weiteren Verwandtschaft nicht vor. Keine Totgeburten. Der ältere Bruder des Patienten ist gehörlos. Keine weiteren Geschwister.

Der heute 16jährige Patient wurde als zweites Kind einer 37jährigen Mutter und eines 39jährigen Vaters geboren. Gravidität und Partus verliefen regelrecht. Als Kleinkind soll er eine Mittelohrentzündung mit eitriger Sekretion aus beiden Ohren durchgemacht haben. Bereits im Kleinkindalter fiel eine Schwerhörigkeit beiderseits auf, die bis zum Tage der Untersuchung keine wesentlichen Schwankungen aufwies.



Abb 1



Abb 2



Abb 3

Abb 1 und 2 Front und Seitenansicht des Patienten zeigen das charakteristische Bild der Dysostosis mandibulo-facialis Hypoplasie der Jochbögen antimongoloide Lidachsenstellung stumpfer Kinnwinkel

Abb 3 Schematisierte Zeichnung des Stapes rechts Der Steigbügel hat die Form eines Knochen saulchens von doppelter Stapeschenkelhöhe Eine Knochenbrücke fixiert ihn an den Facialis kanal Das Ringband ist nur an der vorderen Curvatur des ovalen Fensters angeheftet

Befund (30.6.1959)

Das äußere Bild des Patienten zeigt die charakteristischen Merkmale der Dmf Antimongoloide Lidachsenstellung Aplasie beider Jochbögen Hypoplasie des Unterkiefers mit stumpfem Kinnwinkel (s. Abb. 1 und 2)

Ohren

Äußeres Ohr beiderseits von normaler Form etwas lappig Die äußeren Gehörgänge sind von gehöriger Weite die Trommelfelle grau reizlos intakt im Siegle Trichter gut beweglich Die Tuben sind leicht und seitengleich luftdurchgängig

Die audiometrische Untersuchung ergab das Vorliegen einer links hochgradigen rechts mittel hochgradigen reinen Schalleitungsschwerhörigkeit Gellé'scher Versuch negativ

Nase

Septumdeviation nach links Bodenleiste beiderseits Nasenatmung ausreichend

Nasenrachenraum

Irei kein pathologisches Sekret

Mundrachen

Geiß intact keine Bläsonomalie hoher Gaumen angedeutet Tonsillen mittelgroß

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Knochenbrücke fest mit dem Facialiskanal verbunden (Abb. 3). Nach Durchfräsen der knöchernen Brücke und mehrfacher Umstechung des Knochenkanalchens auf der Fußplatte wurde das Stapesrudiment leicht beweglich und das Gehör auf über 1 m Flustersprache gebessert. Bei der Operation des rechten Ohres, die am 3.3.1960 erfolgte, fand sich die gleiche Form der Mißbildung wie links. Auf Grund des guten funktionellen Ergebnisses, das wir links erreicht hatten, mobilisierten wir auch das rechte Stapesrudiment in der oben angeführten Weise (Abb. 4).

DISKUSSION

Das charakteristische Äußere eines Patienten mit einer Dysostosis mandibulo facialis erlaubt die Diagnose dieser Dysmorphie selbst bei angedeuteten oder atypischen Formen. Umso erstaunlicher ist es, daß dieses Krankheitsbild erst 1944 von Franceschetti & Zwahlen unter Auswertung der bis zu diesem Zeitpunkt beschriebenen Fälle und zweier eigener Patienten als morphologische Einheit betrachtet und als Dmf bezeichnet wurde. Van Lint & Hennebert (1936) hatten ebenso wie Mann (1943) die Originalität dieses Syndroms erkannt, ohne es jedoch als einheitliche Entwicklungsstörung zu isolieren.

Im Gegensatz zu anderen cranio facialem Dysostosen (Crouzon & Apert usw.) findet sich diese kongenitale Mißbildung kaum in den Abhandlungen der Pathologie — nur Virchow (1864) vermutet in einer Arbeit über Mißbildungen am Ohr und im Bereich des ersten Kiemenbogens einen übergeordneten Störungsreis, der den Viszeralbogen einschließt. In diesem Zusammenhang zitiert er die Beschreibung einer Dysmorphie von Thomson (1847), die wir heute als Dmf bezeichnen würden.

Das sehr vielgestaltige Bild der Dmf veranlassen Franceschetti & Zwahlen (1944) die Unterteilung in 1) forme complete, 2) forme incomplete, 3) forme abortive und 4) forme atypique nach Ausprägung der Entwicklungsstörung vorzunehmen.

Bei voller Ausbildung des Symptomkomplexes finden wir bei den geistig meist normal entwickelten Personen folgendes klinische Bild:

1. Antimongoloide Schrägstellung der Augenspalten, Abknickung des unteren Lidrandes im äußeren Drittel und Kolobombildung am unteren, seltener am oberen Lidrand.

2. Hypoplasie der Gesichtsknochen, insbesondere des Jochbeines, des Ober- und Unterkiefers.

3. Mißbildungen des äußeren Ohres.

4. Makrostomie, hoher Gaumen, fehlerhafte Zahnstellung und Bissanomalien.

5. Blind endende Fisteln zwischen Ohr und Mund, Aurikularanhänge.

6. Atypischer Wuchs der Kopfhare.

7. Gelegentliche Vergesellschaftung mit anderen Anomalien wie Gesichtspalten und Mißbildungen des Skelettes.

Vom embryologischen Standpunkt aus handelt es sich um eine Störung der Ossifikation im Bereich des ersten Kiemenbogens obwohl die häufige Deformierung des äußeren Ohres und des Mittelohres auch auf eine Entwicklungsstörung der dorsalen Partie des zweiten Kiemenbogens hinweist. Die Verzögerung des Knochenwachstums tritt zwischen der 7. und 9. Fetalwoche ein. Eine Reihe von Beobachtungen verdeutlichen die Rolle des hereditären Faktors (Berry 1949, Isakowitz 1921, Debusmann 1940, Leopold Mahoney & Price 1945, Straith & Lewis 1949, Franceschetti & Klein 1949). Für den hereditären Charakter der Mißbildung spricht auch das Vorkommen anderer Fehlentwicklungen in der Familie — wie in unserem Falle die Taubheit des Bruders.

Die Entwicklung der Mikrochirurgie des Ohres brachte es mit sich, daß inzwischen schon bei einer Reihe von Patienten mit einer Dmf. die operative Verbesserung des Gehöres versucht wurde. So beschreibt Harrison (1957) 5 Fälle von Dmf. von denen er einen operierte. Er fand in der Pauke rudimentäre Gehörknöchelchen. Spezielle Angaben über den Stapes und besonders über seine Beweglichkeit macht er nicht. Da die Fensterung ein gutes funktionelles Resultat brachte, bestand auch hier vielleicht eine Ankylose im ovalen Fenster. Livingstone (1959) sah unter 20 Patienten, die er wegen Mißbildung des Ohres operierte, 4 Patienten mit einer Dmf. Er berichtet über Mißbildungen von Hammer und Amboß, die er meist an der Paukenwand fixiert und miteinander verbacken fand. Eine detaillierte Beschreibung des Stapes und vor allem die Erwähnung der Prüfung seiner Beweglichkeit fehlt jedoch. Clerc & Sterkers (1959) erwähnen bei der Beschreibung ihrer Mobilisationstechnik einen Patienten mit Dmf. und unbeweglichem Stapes. Die Form des Stapes und die Ursache oder das Ausmaß der Fixation werden nicht dargestellt.

Shambaugh (1952) operierte 22 Ohren mit einer Mißbildung des Mittelohres. Normal war der Steigbügel fixiert, jedoch mit Sicherheit nicht durch otosklerotische Veränderungen im Bereich des ovalen Fensters. Es ist bemerkenswert, daß — ähnlich wie bei unserem Patienten — gerade in diesen Fällen Gehörgang, Trommelfell, Hammer und Amboß von natürlicher Form und Größe waren. Ob außer den Anomalien des Mittelohres noch Fehlentwicklungen des Schädels oder des übrigen Skelettes vorhanden waren, geht aus der Arbeit nicht hervor.

SUMMARY

In a case of *dystostosis mandibulo-facialis* (Treacher Collins syndrome) we found besides the typical deformities of the skull, jaw and eyelids, a marked conductive deafness. External ear, meatus and eardrum were normally formed. In the middle ear we saw a columella-shaped stapes which was fixed in the oval window by malformation of the footplate and a small bony bridge to the facial canal. Malleus and incus had normal size and mobility. Bilateral good hearing result after stapes mobilization.

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MUCO PYOCELL OF SPHENOIDAL SINUS OR POSTERIOR ETHMOIDAL CELLS WITH SPECIAL REFERENCE TO THE APEX ORBITAL SYNDROME

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Almost 60 cases of mucocoele of the sphenoidal and posterior ethmoidal sinuses have been described in the literature. Two new cases are added. The necessity for early diagnosis is emphasized if irreparable damage to one or both eyes is to be prevented. Rhinological examination was informative in one third of the cases. Ophthalmological examination may draw the attention to the apex orbitae. If the examiner is aware of the existence of such a condition thorough radiological investigation including tomography will as a rule give a clear and confident diagnosis. Surgical therapy is best accomplished by endonasal drainage by a transmaxillary approach and not by craniotomy.

If the outlet of an accessory sinus is blocked secretion may accumulate in it with consequent expansion and thinning of one or more of its walls. Such an accumulation of secretion is often bacterial and mucous and the formation is then called a mucocoele. When the contents are purulent it is called a pyocoele. The symptoms and roentgen appearance of a mucocoele do not differ significantly from those of a pyocoele (Larsson 1944, Welin 1951). Gerber (1918) also claimed that mucocoele, pyocoele and empyema with dilatation could produce the same clinical picture. He coined the term *antritis dilatans* as a group name for these lesions. For simplicity, mucocoele and pyocoele will hereinafter be referred to as mucocoele.

Mucocoeles are most common in the frontal sinus and anterior ethmoidal cells and rare in the sphenoidal sinus and posterior ethmoidal cells. According to most authors involvement of the maxillary sinus is always secondary to a mucocoele in some other sinus.

Brown & Goodhull (1956) and others distinguish between primary and secondary mucocoeles of the frontal sinus. In a primary mucocoele a cyst forms from a goblet cell gland and may grow to such an extent as to dilate the sinus.

The secondary mucocoele is caused by obstruction of the sinus outlet by a fracture, a tumour or an inflammatory process. Hence fractures of the nasofrontal duct occasionally cause mucocoeles. The type of tumour that most frequently blocks the outlet of the frontal sinus is osteoma (Howarth

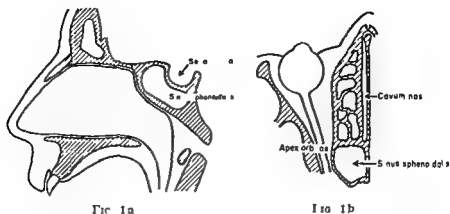


FIG 1 Schematic drawing of sphenoidal sinus and surroundings (a) lateral view (b) axial view

1924 Vidala & Somers 1949) A common cause of mucocoele is chronic sinusitis with obstruction of the outlet of the sinus

The sphenoidal sinus is occasionally affected by fractures of the base of the skull but the outlet of the sinus is exceedingly short as compared with that of the frontal sinus and as far as we know such fractures have never caused a mucocoele of the sphenoidal sinus Furthermore no case of sphenoidal sinus mucocoele due to osteoma has ever been described Chronic inflammation is probably the most common cause of a mucocoele in the sphenoidal sinus

Mucocoele of the sphenoidal sinus was first described in 1889 by the Swedish surgeon John Berg The patient a woman aged 27 was blind and had bilateral exophthalmus bulging of the roof of the nasopharynx and intense headache The headache disappeared after enucleation of the right eye and opening of the mandarin sized sphenoidal sinus which drained a clear fluid via the orbit

Diseases of the sphenoidal sinus are difficult to judge rhinoscopically As a rule acute or chronic infections in this region cause apart from purulent nasal discharge only headache and the possibility of a lesion in this concealed part is not considered until after the appearance of more striking phenomena Proetz (1948) stressed that an inflammatory or expanding benign or malignant process in the sphenoidal sinus can affect a variety of structures The posterior ethmoidal cells are also intimately connected with most of these structures particularly the orbital apex Pathological processes in these cells can therefore produce symptoms simulating those of processes of the sphenoidal sinus (Fig 1) Mucocoeles of the sphenoidal sinuses sometimes affect vessels and nerves passing through the optic foramen and the superior (sphenoidal) orbital fissure with an apex orbital syndrome as a result (syn orbital inlet syndrome sphenoidal fissure syndrome orbital apex syndrome) i.e disturbance of vision paresis of the eye muscles disorders of the sympathetic nervous system exophthalmus and pain The known wide variation in the size of the sinus and the thickness of the walls

(Pendergrass *et al* 1956 1) is sufficient to explain the variety of symptoms and the varying direction of expansion of the mucocoele. According to Proetz (1948) part of the bony walls is sometimes missing.

Fifty three cases of mucocoele of the sphenoidal sinus and posterior ethmoidal cells have been described in the literature to which two may now be added. We have also judged as mucocoeles some cases (Everberg 1955 MacCarty *et al* 1957) which were classified by the respective authors as cysts of the sphenoidal sinus.

Other authors only mention that they have seen such mucocoeles. For example Wiskowsky (1921) briefly cited by Ledinsky & Linhartova (1959) has seen a non expanding mucocoele of the sphenoidal sinus. Bull (1951) only shortly mentions a personal case of sphenoidal mucocoele. R G Lachajev (cit Dobromylskij & Baltin 1952) found among a large number of mucocoeles two cases localized to the sphenoidal sinus and the ethmoidal labyrinth. Dobromylskij & Baltin (1952) presented a personal series consisting of 70 mucocoeles of which four were localized to the sphenoidal sinus. One of these patients was briefly described in the article and is included in the 53 cases mentioned above.

Pendergrass *et al* (1956 3) described a case resembling that presented by Schuller (1932). Already on first examination however the large cyst formation was air filled. The author believed that it was most probably a hypophyseal duct cyst that had emptied spontaneously.

Bordley (1959) in a discussion of Maxwell & Hill's lecture (1959) mentioned that at his clinic he had recently seen two mucocoeles of the sphenoidal sinus with extension into the ethmoidal cells.

PERSONAL CASES

CASE I—J no 1291/58 The patient was a male aged 16 with a history of recurrent infections of the throat, paranasal sinuses and middle ears. On June 1 1958 he had a right sided otitis and meningitis which responded well to antibiotic therapy. Three days later right sided ptosis was observed and examination revealed a temporal defect of the field of vision on the right side. On June 4 light perception was uncertain with temporal pallor of the papilla of the right eye. The symptoms subsided but a defect in the visual field of the right eye persisted. The patient was referred to the Department of Neurology, Lund.

Röntgen examination

On July 22 1958 lateral roentgenograms of the skull showed the sphenoidal sinus to be expanded and extremely dense (Fig. 2). The bony wall between the sella turcica and sphenoidal sinus was rarefied and extremely thin. A P films at an angle of 30° showed the sphenoidal sinus to be expanded also in the transverse plane.

Lateral tomograms of the sella turcica region showed not only the changes described above but possibly also a small bone defect between the sella turcica and sphenoidal sinus (Fig. 3).

Frontal tomograms showed a bulging of the roof of the sphenoidal sinus into the



FIG 6

Ophthalmological examination

The left eye showed no abnormalities. Atrophy of the right optic nerve with poor pupillary reaction to light, and a temporally pale papilla. Temporal loss of the visual field extending over the fixation point. The vision of the right eye was 15/60. Hormone analysis revealed nothing of interest.

Transmaxillary sphenotomy

On August 12, 1958, a right transmaxillary sphenotomy was performed. The posterior part of the ethmoidal bone was removed first and the forwardly bulging, crepitating wall of the sphenoidal sinus was exposed. Resection of the bone revealed a bluish mucosa. Incision of the mucosa was followed by the escape of viscous secretion. The anterior wall was removed and the entire large sphenoidal sinus could be inspected. There were no signs of tumour formation and no dura pulsation.

Postoperative course

This was uneventful. The day after operation the patient noticed that the limitation of the right visual field had become somewhat smaller. Eye examination on various occasions after the operation (last time Feb. 22, 1960) showed no further changes. Check roentgenography (Feb. 22, 1960) showed that the site of previous mucocele was air filled and that contrast medium instilled in the nose readily flowed into the dilated sphenoidal sinus (Fig. 5).

CASE II—J no 1908/58 The patient was a man, aged 53. In 1913 he had acute right sided sinusitis. The ESR was 133 mm/1 hour. Maxillary sinus operation with ethmoidectomy and sphenotomy showed polyps of the sinuses. "The large sphenoidal sinus had three compartments behind one another." Healing was good. During the



FIG. 7

following years sinusitis recurred on various occasions. In 1953, after having a cold for one month the patient complained of double vision and tenderness above the right eye. Investigation suggested minimal paresis of the right abducens and superior rectus muscles. In November, 1957, after having had a non purulent discharge from the nose for a year and severe pain around the right eye for one day, the patient noticed impaired vision on that side which progressed during the next few days. Since then he had had impaired vision and severe right sided headache.

Ophthalmological examination

This showed an amblyopic pupillary reaction on the right side and a temporal pale papilla. Vision 0.1 right—glasses produced no improvement. Central scotoma and possible insufficiency of the abducens and right superior oblique nerves were noticed. The left eye was normal.

Roentgen examination

This was performed on December 2, 1957. Skull and sinuses: lateral views of the cranium showed a density at the site of the sphenoidal sinus (Fig. 6). In the submentovertical projection of the anterior part of the cranium a pear shaped, expanding soft tissue process, widest posteriorly, was seen at the site of the posterior and middle ethmoidal labyrinth on the right side (Fig. 7). Dorsally the sphenoidal sinus was reduced to a narrow slit by pressure of the process on the right side. No septa were seen in the affected part of the ethmoidal labyrinth.

In submentovertical tomograms the changes described were demonstrated still more distinctly than on plain radiograms (Fig. 8).

In P.A. projection the medial orbital wall on the right side appeared to be curved and dislocated laterally (Fig. 9).

Moderate mucosal changes were seen in the maxillary sinus, from where the medial wall had been removed at previous operation. In frontal tomograms the right anterior clinoid process was dislocated cranially and laterally and was somewhat rarefied. Special views of the optic foramina showed that the optic foramen as well as the



Fig 8



Fig 9

superior orbital fissure was markedly dilated on the right side and that the intermediate bone had been destroyed (Fig 10a and b). Lumbar encephalography on December 9, 1957, and right carotid angiography on March 3, 1958, showed no signs of a pathological condition.

During the following year vision of the right eye varied between 1/60 and 0.4. The defect in the field of vision also varied considerably. Right-sided slight ptosis occurred, as well as a very distinct orbicularis oculi spasm and slight downward dislocation of the bulb. Neurological examination showed, in addition to the ocular findings, right-sided anosmia. Otoneurological examination revealed nothing of interest.

In the middle of October, 1958, the headache increased in intensity. Vision had decreased to 1/60 and the central scotoma had become larger.

On roentgen examination of the skull on October 24, 1958, the posterior ethmoidal



Fig 10a



Fig 10b

Fig 10 Case II (a) right orbit (b) left orbit



FIG 11a

cells were found to be denser also on the left side, and the medial orbital wall bulged slightly laterally on that side, too. The right optic foramen was wider than on previous examination. Diagnosis: mucocele of posterior ethmoidal cells on the right side.

Transmaxillary spheno-tomy

On November 21, 1958 the patient was submitted to transmaxillary spheno-tomy. On incision of the ethmoidal region abundant, viscous mucous and yellow fluid escaped. A cyst was found in the ethmoidal region near the sphenoidal sinus. The mandarin sized cyst was opened and a pulsatile flow of similar fluid escaped. The pulsation thus showed that the bony wall facing the cranial cavity had been de-



FIG 11b

FIG 11 Case II (a) lateral view (b) submento vertical view

stroyed. The entire anterior wall was removed and broad connection was established to the maxillary sinus and nasal cavity. Culture of fluid from the mucocoele gave growth of enterococci.

Histological examination of the anterior wall of the mucocoele showed small bone trabeculae and slightly fibrous tissue with diffuse and in some regions fairly scanty deposits of round cells and polymorphonuclear leukocytes.

Postoperative course

An hour or so after the operation the patient reported that the vision on the right side had improved and this was verified by examination on Nov. 21. Vision of the right eye 0.1 (Glasses produced no improvement). Both pupils reacted normally to light. The field of vision on the right side had improved considerably but there was still a central scotoma. The patient was no longer troubled by headache. The orbicularis oculi spasm had diminished. When last seen by the ophthalmologist on Feb. 21, 1960, vision of the right eye had improved to almost 1.0. There was however still a large central scotoma. The downward displacement of the bulb as well as the headache and the ptosis on the right side had disappeared.

Check roentgenography on Feb. 21, 1960. The former mucocoele was air filled and contrast medium instilled into the nose flowed into the sinus (Figs. 11 a and b).

ANALYSIS OF 55 CASES OF MUCO PYOCELE OF SPHENOIDAL SINUS OR POSTERIOR ETHMOIDAL CELLS

Of these 55 cases 36 mucocoeles and 4 pyocoeles originated from the sphenoidal sinus. The corresponding figures for the sphenoidal type were 11 and 1 and for the ethmoidal type 2 and 1.

Age and sex

The patients were aged 13-70 years with a slight predominance of the sixth decade. 29 were females and 24 were males. Sex and age were not mentioned in two cases.

Duration of symptoms

Most patients had had symptoms up to	a few	much
as 20 years before the condition had been		

Headache

Most of them had headache	and
to the forehead or around	the
back of the head was reported	in
and sometimes associated	with
that even when only the	sight
not often localized to the head	and
that pain behind the eyes	was
most common in chronic cases	and
only reported in very severe	

Eye symptoms

Compression of the optic nerves impairs vision and may result in total blindness. This leads to loss or weakening of the pupillary reflexes the optic nerves atrophy and defects appear in the fields of vision. Paresis of the eye muscles are fairly common (20 cases) but sometimes transitory.

	Bilateral	Right	Left
Blindness or very severe impairment of vision	7	5	12
Slightly impaired vision	3	3	5
Exophthalmus	3	7	10

Craniotomy (12 cases) or autopsy (2 cases) revealed that the mucocoele had destroyed the bone and lifted the dura in the area corresponding to the roof of the orbit and sphenoidal sinus. The optic nerve (sometimes both) was lifted and stretched and often atrophic. Extensive destruction of the roof of the sinus and the walls had also been observed in 13 cases at endonasal operations and often seen as pulsations of the walls.

If a mucocoele encroaches upon the orbit exophthalmus occurs and like other eye symptoms and headache this may vary in intensity with the pressure exerted by the expansive process. Symptom free intervals of months or years are not uncommon. A case illustrating fluctuation of the symptoms ophthalmoplegic migraine has been described by Hermann & Hall (1940). They excluded aneurysm by carotid angiography. O'Shea's (1937) patient had a weakly pulsating exophthalmus which was caused by a mucocoele of the sphenoidal sinus.

Nose symptoms

Nose symptoms are sometimes due to the conditions causing mucocoele such as recurrent sinusitis and polyposis. In one fourth of the cases the mucocoele bulges into the nose obstructing the air passage and leads to anosmia or hyposmia. This can also occur when the olfactory organ is directly involved. Occasionally a mucocoele ruptures spontaneously and then the typical contents empty into the nose (O'Shea 1932 Pendergrass *et al.* 1936 2).

The present compilation (33 cases) includes 9 cases with sinusitis in their history. Hajek's (1910) patient had ozæna and Gerber's (1918) and Vessons's (1938) patients had nasal polyposis. Only in some cases then could the mucocoele be ascribed to chronic inflammations with certainty.

Clinical symptoms were often unilateral (14/33). Petit Dutaillis, Thiebaut & Fischgold (1930) stated that a mucocoele of the posterior ethmoidal sinus produces relatively acute and unilateral symptoms while a mucocoele of the sphenoidal sinus is preceded by more protracted symptoms and produces bilateral symptoms. This assertion could not be confirmed on analysis of these 33 cases.

It appears more likely that inter individual variation in the anatomy and thickness of the walls can explain the varying direction of expansion of the mucocoele and why the symptoms are bilateral in some patients and unilateral

stroyed. The entire anterior wall was removed and broad connection was established to the maxillary sinus and nasal cavity. Culture of fluid from the mucocoele gave growth of enterococci.

Histological examination of the anterior wall of the mucocoele showed small bone trabeculae and slightly fibrous tissue with diffuse and in some regions fairly scanty deposits of round cells and polymorphonuclear leukocytes.

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Check roentgenography on Feb. 24, 1960. The former mucocoele was air filled and contrast medium instilled into the nose flowed into the sinus (Fig. 11 *a* and *b*).

ANALYSIS OF 53 CASES OF MUCO PYOCELE OF SPHENOIDAL SINUS OR POSTERIOR ETHMOIDAL CELLS

Of these 53 cases 36 mucocoeles and 4 pyocoeles originated from the sphenoidal sinus. The corresponding figures for the spheno-ethmoidal type were 11 and 1 and for the ethmoidal type 2 and 1.

Age and sex

The patients were aged 13-70 years with a slight predominance of the sixth decade. 29 were females and 24 were males. Sex and age were not mentioned in two cases.

Duration of symptoms

Most patients had had symptoms up to two years and a few up to as much as 20 years before the condition had been recognized.

Headache

Most of them had headache on one side or the pain had been localized to the forehead or around one of the eyes. Diffuse headache or pain in the back of the head was reported by some. The headache was often disabling and sometimes associated with nausea and vomiting. It should be observed that even when only the sphenoidal sinus was involved the headache was not often localized to the back of the head. Maxwell & Hill (1939) reported that pain behind the eyes, in the forehead or the fronto-temporal region is most common in chronic sphenoiditis. Pain in the back of the head was only reported in very severe cases.

Acta otolaryng.

who knows that a mucocele is capable of occurring in this region (Petit Dutailis, Thiehaut & Fischgold, 1950, Fischgold *et al*, 1951, Dobromyskiy & Ballin, 1952)

Roentgenological considerations

When a mucocele develops in a sinus the normally air filled cavity appears as a massive density. As to the sphenoidal sinus, this density was first described by Schuller in a case published by Hajek (1910), and has since been noted in most published cases. When the mucocele grows, it presses against the walls of the sinus, which undergo pressure atrophy resulting in a smooth outlined bony cyst. Meisels (1926) was the first to give a fairly detailed description of these changes and considered them to be characteristic of a mucocele. The bony septa in the sinus involved are destroyed relatively early. When the neighbouring bone is primarily fairly thick, it may be destroyed to such an extent that the sinus appears to be filled with air even though it is in reality filled with fluid. As to the frontal sinus, this phenomenon is well known and has been described by Irostberg (1944), for example. It is uncertain whether this also holds for the sphenoidal sinus because its walls are as a rule relatively thin. Various projections, particularly submento-vertical projections and tomograms, will reveal the true situation. A few mucoceles have emptied spontaneously and then became air filled (Pendergrass *et al*, 1956, 1). With increasing pressure atrophy of the bone surrounding the mucocele, the surrounding osseous canals are roentgenographically widened and the passing structures compressed. Pfeiffer (Bilchick's case, 1940) and later Fischgold (Petit Dutailis *et al*, 1950) and Everberg (1957) described a widening of the optic foramen. Fischgold reported a widening of the superior orbital fissure in the case described together with Petit Dutailis *et al* (1950). This change has occasionally been observed by other authors. Without being directly involved by the mucocele some nearby structures may be displaced. Thus Fischgold (1951) showed that the anterior clinoid process is demonstrated best in frontal tomograms of the skull. The carotid siphon which often touches the lateral wall of the sphenoid sinus, can sometimes be displaced upwards and laterally (Hermann & Hall, 1944). The chiasmatic cistern with its surroundings may also be slightly lifted (Giovine, 1946; Petit Dutailis *et al*, 1958). Litwinowicz (1912) observed that the sphenoidal sinus became clearer after puncture. The diagnosis of mucocele has sometimes been confirmed by endonasal puncture of the mucocele with subsequent injection of thorotrast and radiography, a technique first performed in 1944 by Hermann & Hall. The value of this method has been stressed by Kustner (1952).

To summarise, it may be said that a mucocele is characterized roentgenologically by the fact that the involved sinus becomes dense and gradually expands. Septa in the sinus disappear and the surrounding bone rarefies. Consequent destruction of the bone is always well outlined. The superior orbital fissure is gradually widened, as is the optic foramen. The anterior

clinoid process is sometimes displaced upwards and laterally. In doubtful cases the diagnosis can be confirmed by puncture and injection of contrast medium into the sinus. The carotid siphon is occasionally dislocated laterally, and the chiasmatic cistern may be narrow.

Differential Diagnosis

Mucocele in this position must be differentiated from the following diseases: hypophyseal tumours, craniopharyngioma, meningioma and glioma in the neighbourhood of the sella turcica, intracranial chordoma and cholesteroloma, and finally tumours arising from the nasopharynx, sinus and base of the skull. Pneumosinus dilatans and chronic sphenoiditis may also make differential diagnosis difficult.

Mucoceles of the sphenoidal sinus have often been misdiagnosed roentgenologically as *hypophyseal tumours*. The type of tumour most liable to simulate a mucocele is the chromophobe adenoma which usually enlarges the sella in a forward as well as a backward direction and makes it shallow. It often has a suprasellar component which dislocates the basal cistern and the floor of the third ventricle. When the adenoma grows anteriorly it destroys the bone between the optic foramen and the superior orbital fissure (Lindgren 1954) which can also occur in the presence of an expanding mucocele. In mucocele, however, the entry of the sella is not widened and the floor of the sella, though rarefied, is usually preserved for a long time. If a chromophobe adenoma is left untreated for a long time it may produce such wide spread destruction as to obliterate all characteristics.

The eosinophilic adenoma grows much slower and has a tendency to depress the floor with seeming elongation of the dorsum sellae as a consequence. The eosinophilic adenoma has a tendency to bulge into the sphenoidal sinus but not to fill it completely, and secondary acromegalic changes occur in the cranium.

The basophilic adenoma is as a rule not so large as to affect the sella turcica. *Craniopharyngiomas* are often situated above the sella, frequently calcified and displace the cistern and third ventricle. They may, however, also be situated intrasellarly and occasionally originate from the residual hypophyseal rests in the sphenoidal body, sphenoidal sinus or nasopharynx (Caffey 1950). If a craniopharyngioma is situated in the sphenoidal sinus, preoperative differentiation from mucocele may be difficult if not impossible, particularly if the growth is cystic (Schuller 1952, Pendergrass *et al.* 1956).

Meningiomas and slowly growing *gliomas* in the middle cranial cavity often give rise to pressure atrophy of the surrounding bone, but the sphenoidal sinus is fairly resistant to extrasellar tumours and is usually not involved. In these tumours encephalography shows displacement of the ventricular system and cisterns. Carotid angiography demonstrates deviation of the vessels, and contrast medium often accumulates in the tumours.

Intracranial chordomas which are not common, most frequently originate

from the sphenoccipital synchondroses and sometimes from the nasopharynx. They grow slowly expanding with skeletal destruction of the base of the skull. The centre of these tumours is thus situated further back than a mucocele of the sphenoidal sinus. Chordomas often grow in dorsal direction with compression of the pons and medulla which may be demonstrated by encephalography. They may however also grow anteriorly and parasellarly or down into the nasopharynx. Their capsule is often calcified. Differentiation from a mucocele may on occasion be difficult.

Epidermoids or *cholesteatomas* of the head are relatively rare. They are of two types: intracranial and extradural (growing in diploe). The latter type has a more or less polycystic form and a sclerotic sometimes calcified capsule (Beutel 1939). When the tumour is situated in the frontal sinus differentiation from a mucocele is not possible (Frostberg 1944) and the diagnosis is probably also difficult if the tumour is situated in the sphenoidal sinus.

Miscellaneous tumours of the nasopharynx and sphenoidal sinus and of the posterior ethmoidal cells may cause opacification of the sinus and destruction in the base of the skull. *Malignant tumours* are relatively rare in the sphenoidal sinus but fairly common in the nasopharynx near the foramen ovale and foramen spinosum. These tumours often cause unilateral destruction and grow invasively rather than expansively. Often they have visibly metastasized to the lymph nodes of the neck at the time of the first examination. *Juvenile angiofibromas* grow first in the nasopharynx where they are seen in roentgen films and with posterior rhinoscopy. They may grow into the ethmoidal cells and cause well defined destruction in the base of the skull (Welin 1931). Also *benign polyps* occasionally fill the sphenoidal sinus which then expands as in mucocele (Achslogh & Gottlob 1937 case 2). The differential diagnosis may be difficult and require puncture and injection of contrast medium.

Finally the tumours that are primarily localized to the bone in the base of the skull e.g. *myeloma* or *plasmacytoma* may offer diagnostic difficulties.

Pneumosinus dilatans is a rare disease in which the sinus is expanded as in mucocele but it is filled with air instead of fluid which makes it more translucent (Herrmann 1938). Harrison & Young (1935) consider it to be a mucocele that has emptied spontaneously. Jezegabel (1960) assumes a genetic predisposition to hyperpneumatization while according to Lffenorde (1942) a pneumosinus is caused by an air trapping valve mechanism. The sinus frontalis is usually affected but Bendescu (1932) described a pneumosinus sphenoidalis causing optic atrophy and Brohm (1948) a case localized to the ethmoidal and sphenoidal sinuses.

Chronic sphenoiditis and *posterior ethmoiditis* usually cause bone proliferation and sclerosis but seldom bone destruction. Sclerosis in this localization may occasionally be seen in a metastatic growth of an osteoplastic carcinoma but in such cases there are usually widespread osseous changes.

Treatment

Because of wrong preoperative diagnosis craniotomy + puncture was the first surgical procedure in as many as 13 cases with surgical establishment of a permanent endonasal drainage in 7. In 13 patients when the mucocele bulged into the orbit or nasopharynx the lesion was drained to the nose. Ethmoidectomy and sphenotomy had been done endonasally in 13 cases and via an external approach in 10. Transseptal sphenotomy had been performed in 7 cases. Our 2 cases were treated with transmaxillary sphenotomy.

Course after Operation

Headache, double vision and nose symptoms disappeared in almost every operated case. In most cases exophthalmus disappears but in some long standing ones a certain degree of exophthalmus persisted.

Patients with double sided blindness had usually not recovered vision and only a few have experienced unilateral improvement. Half of the unilaterally blind patients have not regained vision but the other half usually with short standing blindness have regained normal vision. When vision had been only partly impaired the patient usually recovered original vision. Our own two cases show that permanent blindness or severely impaired vision can result even after only a few days of symptoms and also that a severe impairment of a year's duration can disappear.

Three fatal cases of mucocele have been described after craniotomy. In a case Lindholm *et al* (1946) the patient died one and a half years after operation (see p 612). In 1 of the 3 cases of Vincent *et al* (1946) craniotomy revealed a mucocele which was punctured and the symptoms disappeared. The operating surgeon warned against craniotomy in case of recurrence and recommended endonasal operation instead. While abroad the patient had a recurrence and a craniotomy was done but the patient died. The case of Ledinsky & Lindholm (1949) ended fatally in meningitis 15 days after the operation.

ZUSAMMENFASSUNG

Bis heute sind in der Literatur ungefähr 60 Fälle von Mucocelen der Keilbeinhöhle und hinteren Siebelseiten beschrieben. Diesen werden zwei neue eigene Fälle hinzugefügt. Dabei wird vor allem zur Verhütung von Dauerschäden in einem oder beiden Augen auf die Frühdiagnose der Affektion hingewiesen. Die rhinologische Untersuchung erlaubt die Diagnosestellung in einem Drittel der Fälle. Diagnostisch-morphologische Abklärung kann die Aufmerksamkeit auf die Veränderungen in der Maxillaris richten. Wenn der untersuchende Arzt an die Möglichkeit einer Mucocèle denkt, so gilt dies zwingend inklusive Tomographie eine zuverlässige Diagnose. Die Therapie der Mucocèle ist eine chirurgische, am besten durch endonasale transmaxilläre Drainage aber nicht durch Craniotomie.

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VESTIBULAR REACTIONS DURING PREORBITAL FLIGHT

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One can presume which additional forces will effect the astronaut placed in the nose cone. The greater part of these stimuli will consist of linear accelerations and one can therefore predict effects on the g receptors with resulting singular sensations. With the aid of special arrangements in a human centrifuge, aiming at changing the resultant between linear acceleration (centrifugal force) and gravity force, the resulting sensations during a number of dynamic conditions have been studied. By applying Graybiel's law of the otoliths, it seems permissible to draw some conclusions as to the probable vestibular experiences of the astronaut, at least during the gravitational part of the trip. However, the period of weightlessness during this phase still remains unsufficiently known. The character of the vestibular sensations added to the lack of external visual references will considerably aggravate the difficulties for an active intervention by the astronaut in cases of incorrectness of the flight path.

In space projects in the near future a human being in a rocket driven vehicle will be hurtted out into space describe a wide curve under sub- or zero gravity conditions and then again return to earth. During the major part of the journey the pilot will have limited chances of orienting himself visually with the aid of a periscope. Even if he is able to look out he will miss well known visual references (horizon sun etc.). With certain differences he will find himself in the same situation as a pilot in an airplane who is partly or totally restricted to drawing information from his instruments. These differences can be expressed as follows: on the one hand he will not have the advantage of experience and on the other he will be passively exposed to the accelerations applied.

On the whole he will be concerned with linear rather than angular accelerations and from the vestibular point of view therefore his sensations will derive largely from stimulations of the so called g receptors, that is to say the otoliths, proprioceptors and exteroceptors.

Just like the pilot in his airplane the astronaut in the nose cone will get his positional information from two sources: the instruments and his sense of equilibrium. Graybiel (6) has stated a "law of the otoliths" which demonstrates how the g receptors react when a person is subjected to accelerative

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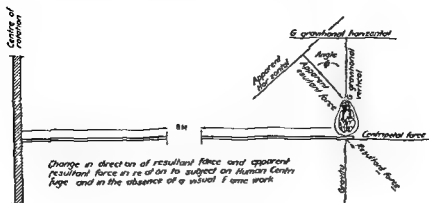


FIG. 1 The law of the otoliths. See text

forces in the absence of visual cues. This law is illustrated by Fig. 1 which shows that the perception of vertical down will eventually coincide with the direction of the resultant of acting accelerative forces and the force of gravity.

Using special test equipment in a human centrifuge I have by a system of remote recording been able to measure the apparent deviations of the vertical and simultaneously vary the direction of the resultant in relation to the test subject.

By essentially reproducing the probable dynamic conditions during different phases of the rocket's path it has been possible to form a rough opinion of the astronaut's vestibular reactions (illusions). It has been assumed that the rocket does not turn about its axis and that its inhabitant the whole time has his head directed outwards.

Probable dynamic conditions during the flight (Fig. 2)

Firstly at the start of a flight the astronaut is subjected to a progressively increasing linear acceleration causing forces to act on him in the same direction as those due to gravity. This acceleration increases as the atmosphere grows thinner. After a few minutes the rocket deviates from its strictly vertical track and a centrifugal force appears which is directed outwards and at right angles towards the tangent of the direction of motion. Then the capsule is released and continues its trip alone in space. The propulsing acceleration ceases at the same time as the centrifugal force becomes more and more accentuated. It will counteract gravity which means that the man in the nose cone will experience weightlessness. The duration of this condition will vary according to the duration of the flight.

As the capsule rushes on it turns 180° resulting in the astronaut being placed with his back towards the direction of motion. This turn probably takes place relatively slowly and does not involve any marked effect on the semi-circular canals.

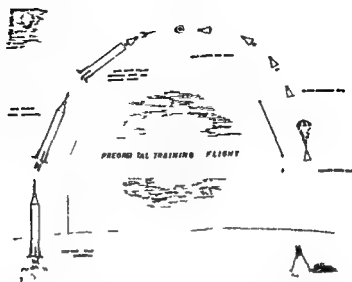


FIG. 2 Schematic description of the trajectory. The following abbreviations are used: GF direction of gravity force; ADG apparent direction of gravity; AI direction of propulsive acceleration force; AT direction of acceleration inertia force; CI direction of centrifugal force; DP direction of deceleration force; DT direction of deceleration inertia force.

At a certain time the retro rocket is fired and a strong deceleration is imposed on the capsule and its inhabitant, producing a new dynamic condition. From this moment the capsule starts its downward trip. A centrifugal force again begins to be felt until the point when the motion of the capsule comes entirely under the control of gravity. Now it becomes a purely vertical drop which is broken by contact with the denser layers of the atmosphere. There arises a sudden deceleration force acting in the same direction as that due to gravity. During this step presumably the most problematic of the trip especially complicated dynamic conditions arise, definitely very stressful for the astronaut.

METHOD

In an earlier paper (2), I gave an account of an apparatus for remote recording of horizontal and vertical perception in the centrifuge. The construction of the apparatus, which apart from a few modifications, does not differ essentially from that described by Graybiel (4) is as follows.

The subject is seated in a cockpit seat on the horizontal centrifuge platform with his head and body fixed rigidly. External visual cues are excluded by means of a shielding box with the test subject in complete darkness. Various devices allow the seat to be rotated slowly around its axis as well as tilted sideways, forwards and backwards to produce the greatest possible variations in the *relative* direction of the resultant to the head. At a normal reading distance in front of him the subject is shown a slightly luminous line, move

able around its centre against a dark ungraded background. This line can be rotated or elevated by pushing and pulling a lever. The setting of the line can be read off in degrees in the control room by means of a system of Selwyn repeater motors over the slip rings of the centrifuge. A corresponding system allows the setting to be offset in the control room by the observer. At the same time it is possible to make continuous recordings of the swing of a pendulum at the level of the subject's head i.e. the direction of the resultant.

If the subject sits facing the direction of turn of the centrifuge acceleration gives an illusion that the line rotates *clockwise*. If he sits facing the centre of the centrifuge he has an illusion that the line rises etc.

During each trial the subject was required to adjust the luminous line from an offset position to what seemed to him to be the vertical i.e. the apparent direction of gravity. The tests were divided into five different series. In each series an attempt was made to simulate as closely as possible the calculated dynamic situation during different stages of the trip. This was carried out in the following manner. On running the centrifuge at a preset rpm a certain alignment of the moveable chair was chosen. Combining both these variable procedures it was possible to produce in each test run the *same direction* of the resultant force in relation to the head of the subject as could be *expected* in each of the different stages of flight. This was achieved through the variation of both the position of the chair and the direction of the resultant.

A predetermined rpm was chosen for the centrifuge correlating to a definite direction of the resultant after which the chair was aligned in such a direction that the head of the test subject was influenced by this force at the same angle of incidence as during any of the situations described below. Immediately before each trial with the centrifuge stationary the test subject was required to adjust his luminous line ten times from an offset position to what seemed to be the vertical position.

After this the centrifuge was run at the predetermined rpm and the procedure was repeated in the same manner. The mean values of the recorded readings in the control room afforded finally a measure of the apparent direction of gravity and its angle of deviation from the direction of gravity.

It is not my intention to load this paper with tables of figures and calculations but as a conclusion it may be stated that the apparent direction of gravity in all cases was in all practical respects identical with the direction of the resultant force.

Probable vestibular reactions

Force diagrams have been drawn on the left in the following figures. It must be pointed out that the symbols are not proportional.

To the right is presented on each figure a corresponding diagram as to the sensations derived from experience in the centrifuge. Apparent direction of gravity (ADG) is the resultant obtained by vectorially compiling different and

FIRST PHASE OF ROCKET ACCELERATION

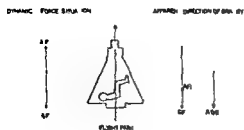


FIG. 3

LATER PHASE OF ROCKET ACCELERATION

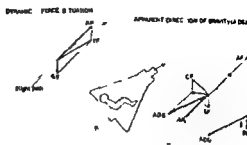


FIG. 4

FIG. 3 First phase of rocket acceleration No orientation problem

FIG. 4 Later phase of rocket acceleration Centrifugal force setting in. Below to the right apparent deviation between objective and subjective vertical

simultaneously acting contact forces due to rocket acceleration or deceleration the centrifugal force when it sets in and the force of gravity.

At launch (Fig. 3) the linear acceleration prevails having the same direction as the apparent acceleration of gravity. Consequently the astronaut is adequately oriented.

In the next stage (Fig. 4) the rocket increasingly diverges inwards from its vertical track. Therefore a centrifugal force (CF) arises, first very faint, then after more and more pronounced. If we choose a point of the path during this phase, one can imagine the dynamic condition indicated on the figure and the corresponding sensorial diagram. We see that ADG becomes approximately tangential toward the flight path.

As a rule one can consequently say that during the whole period of acceleration the astronaut never has the feeling that his course becomes increasingly horizontal. On the contrary, he is convinced that he stays in a vertical direction of motion.

During the third period, when the capsule is released from the rocket, the forward driving force of acceleration naturally ceases. From this moment the centrifugal force will counteract gravity and sub- or zero gravity ensues. Hitherto the astronaut has had his space orientation anchored to a force resultant with a decided direction. Then all linear stimuli suddenly cease.

How a relatively prolonged state of non appearance of linear accelerative stimulus is experienced naturally cannot be predicted, as too little is known of what happens when the receptors of the inner ear entirely lose the constant stimulus of gravity. In spite of the lack of supporting proof for this assumption it may well be questioned if the mystery which surrounds the question of zero gravity, at least for a short period, will not result in a relatively speedy adaptation where vision and exteroceptors (in skin and associated tissues) swiftly step in as compensating impulse producing organs. In the absence of accelerative stimuli there will above all be these organs which play the determining role in orientation. It will consequently seem quite

direction of gravity. In this stage, the astronaut has plenty of outer visual references, making it easy to recognize the horizon.

In this rather brief and naturally very schematic analysis, I have tried to point out in concrete form some of the psycho-physiological problems which the astronaut will meet with in the very marked vestibular reactions to which he will be exposed during his trip in space. It can be assumed that, in connection with violently changing linear vestibular stimuli, especially during the strong initial acceleration and the deceleration caused by the firing of the retro-rocket and the contact with the layers of the atmosphere, he will be subjected to motion sickness. Such subjective disturbances were also noted during the above mentioned experiments in varying degrees. Under real conditions it is assumed that neuro-vegetative disturbances of a similar nature on the whole might be synchronized with other vascular phenomena, such as black-out.

RÉSUMÉ

On peut présumer des forces additionnelles agissant sur le pilote installé dans la pointe du projectile. Ces stimuli consistant en majeure partie en accélérations linéaires, on devra s'attendre à un effet sur les récepteurs g donnant lieu à des sensations singulières. Par le moyen d'un appareillage approprié dans la centrifugeuse humaine, visant à varier la résultante entre l'accélération linéaire (force centrifugale) et l'accélération terrestre, on a analysé les sensations produites lors de différentes situations dynamiques.

En appliquant la "loi des otolithes", énoncée par Graybiel, il serait possible de tirer certaines conclusions de probabilité quant aux expériences vestibulaires de l'astronaute. Ces conclusions s'appliqueront plutôt à la phase "gravitationnelle" du trajectoire. Pour la période de zéro-gravité, la question demeure toujours dans le domaine des suppositions vu le manque d'informations à ce sujet.

Ces sensations vestibulaires ajoutées au manque quasi total d'informations visuelles externes augmenteront considérablement les difficultés d'une intervention active de la part de l'astronaute en cas de "fausse route".

ZUSAMMENFASSUNG

Man kann sich vorstellen, welche zusätzlichen Kräfte auf den in der Raketenspitze platzierten Astronauten einwirken werden. Der grössere Teil dieser Stimuli wird aus linearen Beschleunigungen bestehen und man kann daher die Einwirkungen auf die g Rezeptoren mit resultierenden Einzelempfindungen voraussagen. Mit Hilfe von besonderen Anordnungen in einer Menschen Zentrifuge, mit dem Ziel einer Veränderung der Resultante zwischen linearer Beschleunigung (Zentrifugalkraft) und Schwerkraft, hat man die resultierenden Empfindungen unter verschiedenen dynamischen Bedingungen studiert. Bei Anwendung von Graybiel's „Otolithen-Gesetz“ scheint es zulässig, einige Schlussfolgerungen betreffend die wahrscheinlichen vestibulären Ergebnisse des Astronauten zu ziehen, zumindest während des „Schwerkraft“ Teiles der Fahrt. Die Periode der Gewichtlosigkeit während dieser

Phase verbleibt jedoch noch immer ungenugend bekannt. Der Charakter der vestibulären Sensationen, zusammen mit dem Fehlen von ausseren visuellen Haltepunkten, wird die Schwierigkeiten für ein aktives Eingreifen des Astronauten bei Ungenauigkeiten in der Flugrichtung bedeutend erhöhen.

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Sylvesterbogen 3 A, Lidingö 1

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THEODOR DEMETRIADES

In Memoriam

Von seinem schweren Leiden erlost, verstarb Ende Juli 1960 in seiner Heimatstadt der o Prof Th Demetriades der Universität Athen nach langwieriger Krankheit Theodor Demetriades wurde als Sohn des Univ Prof Dr St Demetriades, der ebenfalls Oto Rhino Laryngologe war, in Athen geboren, besuchte dort das Gymnasium und nach Beendigung desselben inskribierte er sich an der Athener Medizinischen Fakultät und nach Absolvierung letzterer rückte er im ersten Weltkrieg ein als auch im Griechisch Türkischen und schliesslich im letzten Weltkrieg wo er wieder nochmals als Sanitätsoffizier diente

Seine erste Fachausbildung genoss er bei seinem Vater, dann kam er im Jahre 1923 nach Wien, wo er an den Kliniken Alexander, Hajek und Neumann seine Fachausbildung vollendete An der letzten habilitierte er sich als Privatdozent im Jahre 1927 Im Jahre 1930 übernahm er die Oto Rhino Laryngologische Abteilung des Roten Kreuz Krankenhauses in Athen und im Jahre 1939 wurde er zum Professor an der Athener Universität ernannt, wo er von Anfang an einer der führenden Oto Rhino Laryngologen war

Seine zahlreichen wissenschaftlichen Arbeiten beziehen sich hauptsächlich auf die Otologie, aber auch in den anderen Abschnitten unseres Fachgebietes hat er vieles publiziert

G E Iannoulis

Österreichischer Oto Laryngologentag 1960, Zell am See, 14 bis 16 Oktober

In Zell am See stand der 1. Tag im Zeichen der Vorträge über die Schadelbasisfraktur *Hibler* (Ischl) hat zusammen mit *Kruger* vorgeschlagen, bei frontobasalen Frakturen und ausgedehnten Duraverletzungen bei der Operation mit Muskellappen den sonst offenen Zugang zur Nase abzutamponieren, um eine aufsteigende Infektion zu vermeiden. In der Diskussion haben sich auch *Zange*, *Unterberger* u. a. für dieses Vorgehen eingesetzt.

E. H. Mayer hat sich bemüht, auf Grund der beobachteten Zwischenfälle bei Tonsillenoperationen auf die Möglichkeit der Vermeidung hinzuweisen. Besonderer Wert wird dabei auf die entsprechende psychische und medikamentöse Vorbereitung gelegt, Aufnahme am Vortag vor der Operation, Vorbereitung am Abend mit Luminal bzw. Nembutal, vor der Operation Barbiturate und jedenfalls Atropin, auch bei Kindern Narkose jedenfalls am liegenden Patienten. Es wurden bei Tonsillenoperationen in Lokalanästhesie bei der Durcharbeitung des Wiener Materials einige Fälle (20% der Todesfälle, insgesamt 3 Todesfälle in 15 Jahren) mit reflektorischem Herzstillstand gefunden. Unabhängig von der Art der angewendeten Anästhesie handelte es sich in 25% der Todesfälle in Wien in den letzten 15 Jahren um Peritonissilarabszesse, Abszessotomien, also septische Prozesse. Bei diesen Fällen wird es mit der Intratrachealnarkose wohl möglich sein, die nötigen Eingriffe gefahrloser durchzuführen. Einige Zwischenfälle waren durch Nachnarkotisieren (Äther) und anschließende Aspiration bedingt. Falls wegen starker Blutung und hochgradiger Unruhe des Kindes die Fortsetzung der Operation bzw. Blutstillung nicht möglich ist, kommt auch hier nur die Intratrachealnarkose in Frage.

Richler aus der Abteilung *E. H. Mayer* hat gemeinsam mit *Kellner* über die Gefäßentwicklung des Innenohres berichtet. Es handelt sich um Totalserien von Embryonen von der 4 bis 16. Woche. Ebenso wie die Arteria stapedia die Blockbildung zwischen den Stapesschenkeln verhindert, ist die Vena und Arteria canaliculocohleae der Platzhalter für das runde Fenster.

BOOK REVIEWS

BOENNINGSHAUS, H. G. *Die Behandlung der Schädelbasisbrüche* Georg Thieme Verlag, Stuttgart 200 Seiten

The author first reviews the literature, from the report presented by Voss in 1909 up to the present day (1959). He has collected 175 cases of fracture of the base of the skull, not the result of shots or war injuries but due to traffic and industrial accidents amongst civilians. The fractures are divided into fronto basal and latero basal. By means of a clear and systematic arrangement of the series into a large number of chapters, the author gives us a very instructive picture of the diagnosis and treatment of these injuries and their resultant complications and functional disorders. The book is intended primarily for oto rhinologists who seldom encounter these particular injuries resulting from accidents, but such a comprehensive and clear monograph as this one is bound to be of great value and interest to any department of oto rhinology.

In the section on the roentgen diagnosis of fractures through the nasal sinuses the author states that "die auf dem Roentgenbild sichtbaren Frakturen mit den bei der operativen Versorgung aufgedeckten vergleicht, recht häufig ausgedehnter und verzweigter sind, als man vor der Operation auf Grund des Roentgenbildes angenommen hat" but he does not emphasize that tomography is often of value. Neither does he mention that in all fractures through the frontal sinus, operative exposure must be followed by examination of the posterior wall where one may often find fractures which were not perceptible on the roentgenogram and which, in a large percentage of cases, have damaged the dura and therefore entail a danger of Spat Meningitis.

On the basis of my own experience I would like to classify all fronto basal skull fractures which penetrate the frontal sinus or the roof of the ethmoid as belonging to a group of injuries which constitutes an imperative indication for operation.

The list of references is extremely comprehensive and probably so complete that it will be a great help to anyone wishing to carry out more detailed research on these problems.

Although the author says that no war injuries were included in the analysis, it seems that a certain amount of wartime experience has influenced both the evaluation of the indications and the technique of operation. As the author very rightly points out in his introduction, Professor Mittermaier, who has wide experience in this field, must have contributed to the recommendations made in the book.

Paul Frenckner

KIMIKAE, ISCHIRO *The Structure and Function of the Middle Ear* The University of Tokyo Press, Japan Publications Trading Co., Tokyo 157 pages

In Chapter I the author presents a comparative anatomic study of the middle ear in vertebrates. This consists mainly of a comprehensive summary of factors known earlier, but there are also some tables of the resonance frequency of the external meatus and comparison of the area surrounded by the tympanic membranous ring with the area of the oval window and other comparative anatomic studies which

may be considered of importance to our understanding of the functions of the human ear

Chapter II not only describes the known facts regarding the anatomy and function of the tympanic membrane but also reports a large number of the author's own studies on surface measurements, structure, thickness, shape, etc., and also stroboscopic and other examinations demonstrating the movements of the tympanic membrane. His results are on the whole the same as those of earlier investigators, which are thereby convincingly confirmed.

Chapter III provides a particularly interesting picture of the appearance and function of the auditory ossicles. The author has availed himself of the most modern aids in his investigations and has demonstrated in a large series of studies variations and ranges of variations.

Chapter IV, which deals with acoustic reflexes and their effects on the sound-conducting apparatus, is the most experimental part of the book and gives a good idea, not only of the fine equipment used and the enormous amount of work behind the results, but also of the author's great acoustic and physiologic knowledge.

The author has employed all the latest scientific aids in electrophysics, electron microscopy, acoustics, etc., and with these, combined with a tremendous amount of hard work, has perhaps advanced a step further than his predecessors, at least in some fields.

The book is altogether very interestingly written for the otologist who wants a detailed picture of the subject, but it will certainly also be of great interest to the research worker, who will find here a comprehensive list of references, as well as valuable details resulting from the author's own careful investigations.

Paul Frenckner

HILLY, A. *Atlas der Bronchoskopie*. Georg Thieme Verlag, Stuttgart. 96 pages, 180 Ill.

The author is at the same time head of the department of pulmonary surgery and head bronchoscopist at a German sanatorium, with the result that he has himself several times been able to verify his bronchoscopic findings at operation.

In the introduction the author describes the technique of bronchoscopy and its indications and contraindications. Among the contraindications for bronchoscopy he mentions the occasions when the lesion is so extensive that one cannot hope to cure the patient, and cases in which other incurable diseases coexist. The former contraindication should not be accepted too generally, such an extensive change in the lung ought not in itself to constitute a contraindication for bronchoscopy. The diagnosis based on bronchoscopy and biopsy of a specimen may possibly be a guide to treatment, as, for example, in oat cell sarcoma, where very considerable changes in the lungs may regress after radiation treatment. The introduction also contains illustrations and designations of the different bronchi.

The author later gives 22 pages of general description of the normal and diseased bronchial tree, referring to various bronchoscopic photographs forming a separate section of the book. Out of 1000 photographs, the author has chosen 180, most of them in colour, showing normal conditions, mechanical and inflammatory changes, different kinds of tumours and, further, some pictures taken before and after resection of biopsy samples or operation. Alongside each illustration there is a short

L. RÜEDI *Fortschritte der Hals Nasen Ohrenheilkunde Part VI* S. Karger AG Verlag Basel

This part reports the treatment and pathology of malignant tumours of the upper jaw, the results of treatment of such tumours of the larynx and hypopharynx and different methods of treating corrosive damage to the oesophagus

The paper on the treatment of malignant tumours of the upper jaw is written in French by Pietrantonio. One hundred and thirteen operation cases were followed up. The results achieved by the combination of operation and cobalt treatment (28 % five year healing) were better than those with surgical and roentgenological methods where there was 23 % five year healing.

From the Department of Otology at the University of Zurich Suter reports 198 cases of tumours of the larynx and hypopharynx, including 107 of the larynx operated upon at the department. The different stages of these are described statistically and comprehensive tables are given. Laryngectomy was carried out in 86 cases. The writer recommends neck dissection in the majority of cases followed by repeated check ups during the first three to four years. Regional lymph node metastases were present in 4.6 % of cases of laryngeal tumour, in 50 % of cases of tumour of the hypopharynx and in 40 % of cases of vallecular tumour. There were recurrences or remote metastases in 17 % of laryngeal tumour cases and in 43 % of cases of hypopharyngeal tumour. In some cases roentgen therapy was given after the operation. Eighty six per cent three year healing and 82 % five year healing was noted in cases of laryngeal tumour operated by the author. The corresponding figures for tumours of the hypopharynx were 47 % and 47 %.

Becker & Haas give an account of chemotherapy in cases of malignant tumours of the ear nose and throat. Briefly summarized nothing has yet been found which helps.

Burian from Vienna has contributed a detailed and extremely interesting paper on the treatment of corrosive damage to the oesophagus giving a survey of earlier papers on the subject followed by his own series. He advocates the immediate administration of cortisone (prednisolon) in all cases of corrosive damage to the oesophagus even when the damage is only suspected. The doses must be large up to 100 mg prednisolon every 24 hours for an adult. Five to six days after the initial damage oesophagoscopy should be carried out under anaesthesia and the amount of cortisone to be taken decided on the basis of the findings. Subsequently a further inspection should be made each week. In most cases the passage of a sound can and should be avoided and is only resorted to if granulation tissue forms. Complications are comparatively rare and fewer than with earlier methods and the results are very good. On the other hand cortisone does not help against old cicatricial strictures and in such cases a sound must be passed.

B. G. Olsson

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COMPTE RENDU
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PADOUE, LE 28-31 AOÛT 1966

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LES GRANDES DÉCOUVERTES DANS L'OREILLE ET
DANS LE LARYNX PAR LES ANATOMISTES DE L'UNIVERSITÉ DE
PADOUE DU XVI^e SIÈCLE

Allocution de Président

MICHEL ARSLAN

Les organes des sens ainsi que la physiologie des sensations dans la médecine du Moyen Âge et même dans la philosophie aristotélique du XVI^e siècle étaient une espèce de *no man's land*. On affirmait qu'une partie de l'âme circulait dans les yeux, les oreilles, les organes du goût et du tact. Les éclectes de Galién appelaient *aer innatus* « cette atmosphère » qui dès qu'elle était touchée par un rayon de lumière ou une onde sonore produisait des vibrations. Ces vibrations étaient considérées comme tout à fait métaphysiques. Et même la connaissance exacte de la forme de ces organes n'aurait jamais pu expliquer la façon dans laquelle ces vibrations se produisent.

Pour cette raison, les anatomistes du Moyen Âge et des XVI^e et XVII^e siècles ne considéraient d'aucune utilité la dissection des organes des sens : cela n'aurait rien expliqué de la fonction sensorielle, les différentes parties anatomiques (cartilages, membranes, liquides du bulbe oculaire, épanchements nerveux) n'étant qu'une bizarre disposition et ces nœux et sans aucune finalité de la matière dont était composé le corps humain.

L'école de philosophie qui dans la première moitié du XVI^e siècle adversait les aristotéliques, une école dont le chef était le grand Copernic, avait commencé à dégager la pensée humaine de l'immobilité dogmatique de ces affirmations : ces philosophes proclamaient en effet que les organes des sens ne sont pas comme le voulaient Aristote et la philosophie du Moyen Âge de simples instruments de l'âme, mais ils sont comme des canaux, en certains sens des voies d'accès à l'âme, qui n'a pas son siège dans les yeux, dans les oreilles, mais qui est situé plus profondément et qui était « modifiée » par les agents extérieurs (la lumière, les sons).

Voilà déjà esquisse le fait fondamental de la physiologie sensorielle : c'est à dire que les stimulants physiques qui viennent de l'extérieur subissent une transformation des qu'ils sont en contact avec les organes spécifiques.

Le barrage que la pensée aristotélique de ce temps-là opposait à la recherche anatomique consacrée aux organes des sens explique la situation très singulière de la deuxième moitié du XVI^e siècle. Dans cette période, les grands systèmes du corps humain, tels que l'appareil de locomotion, les grands organes de la cavité thoracique et abdominale, le système cardiocirculatoire, avaient été décrits avec une grande exactitude et avec une grande finesse : les anatomistes avaient même réussi à esquisser dans leurs lignes générales la physiologie de ces viscères. Par contre, un silence presque absolu dominait sur ces parties du corps humain qui servent à la vie de relation.

En effet la doctrine Galénique de la médecine était la seule à être admise parmi les enseignements officiels. L'absolutisme idéologique et philosophique excluait toute possibilité d'appliquer la méthode de dissection anatomique qui avait donné des résultats si importants dans le domaine des viscères et l'étude des instruments de l'âme c'est à dire des organes de sens.

Mais en 1537 le Conseil des Doges appelle à Padoue le très jeune anatomiste de Bruxelles André Vésale et lui confère la chaire de chirurgie en l'obligeant à enseigner en même temps l'anatomie. Vésale n'a que 23 ans. Il arrive ici précédé d'une réputation déjà grande. Il avait étudié à Louvain et à Paris. Anatomiste passionné, il bravait toutes les répugnances et tous les dangers pour chercher les secrets de la vie dans le corps décomposé.

Mais c'est à Padoue seulement dans cet ardent foyer de pensées claires et d'actions énergiques — comme on exprime O. Bichard — que le grand novateur pouvait s'exprimer avec une audace encore mal recueillie à Louvain dans son pays natal et à Paris qui était en quelque sorte la forteresse du galénisme. Cela ne veut pas dire que son enseignement fut paisible car nul plus que lui n'eut à se défendre et nul ne montra plus d'ardeur polémique. Tout en affectant d'admirer Galien dans l'Université de Padoue qui était la première université du monde qui permettait une libre critique au Galénisme continuait en même temps à maintenir la chaire où l'on enseignait la doctrine officielle du grand génie de Pergame. Vésale relevait les multiples erreurs du galénisme et les contredisait sans cesse.

Il serait long de dresser le catalogue de ses découvertes parmi lesquelles je dois souligner l'appareil moteur de l'œil, la position du cristallin dans le globe oculaire, certaines structures de l'oreille, les arthénoides. Son œuvre est réellement un monument de génie et de pouvoir d'observation.

André Vésale quitte Padoue à l'âge de 28 ans après avoir fondé l'anatomie poursuivie avec une méthode strictement scientifique et moderne. Son œuvre « *De humani corporis fabrica* » est imprimée dans la même année 1544 de l'apparition du chef d'œuvre de Copernic, le grand réformateur de la physique aristotélique. Coïncidence singulière qui unit ces deux génies initiateurs de l'ère scientifique moderne.

Les successeurs de Vésale à Padoue s'appellent Realdo Colombo, Gabriel Falloppius, Fabricius d'Acquapendente, Jules Casserius. C'est à ce groupe de savants qu'on doit l'édification de l'anatomie des organes des sens qui forme un « *corpus doctrinae* » parfaitement valable même aujourd'hui. Ils ont aussi posé les premières pierres de la physiologie. Pendant presque cinquante ans ils décrivent dans l'homme et dans les animaux la structure des organes sensoriels avec une finesse et une précision que nous essaierons en vain de retrouver dans les travaux d'aujourd'hui.

Il faut rappeler que toute observation conduite par les chercheurs de ce temps là était réalisée suivant deux principes fondamentaux que la science d'aujourd'hui hélas s'est contrainte de laisser de côté. Le premier est de maintenir dans le groupement des faits et dans leur description une vision toujours unifiée de l'observation anatomique, ce qui fut que chaque partie

d'un organe est toujours considérée comme faisant partie d'une unité biologique c'est pour cette raison que Casserius par exemple ne se limite pas à nous donner la description des osselets humains mais il les compare aux osselets des mammifères et des autres espèces animales en anticipant ainsi l'anatomie comparée le deuxième principe suivi par les grandes anatomistes du XVI^e siècle est la exigence artistique dans le dessin anatomique On ne concevait pas alors que l'art puisse rester séparée de la science la reproduction d'une forme quelconque faite par la main de l'homme devait obéir à l'inspiration et aux strictes règles artistiques C'est ainsi que les « *Tabulae anatomicae* » et la « *De humani corporis fabrica* » de Vésale ont été peintes par un élève flamand de Titien van Calcar un véritable chef d'œuvre de la peinture de la Renaissance

L'anatomie et la physiologie des organes des sens en particulier de l'oreille et de l'œil naît donc à Padoue et s'épanouit avec une extraordinaire vitalité Entre 1561 et 1612 apparaissent six livres dans des éditions somptueuses et superbes qui sont dédiés aux organes sensoriels et au larynx

En effet le larynx était aussi considéré dans la médecine de ce temps un organe étroitement lié à l'âme pour cette raison les anatomistes le décrivent dans les traités d'anatomie « sensorielle » et c'est Fabrici qui de ses études sur le larynx de l'homme et des animaux tira la première théorie vraiment scientifique sur le mécanisme du langage Dans aucune autre période de l'histoire de la médecine il n'y a une floraison si puissante de publications sur les appareils qui assurent les relations entre le monde extérieur et le monde somatique et psychique

L'explication la plus simple de l'acharnement avec lequel ces savants fouillent dans les mystères des petits organes tels que l'œil et l'oreille (et dans lesquels toute sorte de tissus sont présents) avec une finesse et une perfection structurale qu'on ne rencontre dans aucune autre partie de l'organisme se dégage du développement des sciences physiques qui s'avèrent dans ce même siècle Libre de la coercition aristotélique poussé à observer d'un œil attentif la nature fier des découvertes géographiques et des grands voyages accomplis par les navigateurs de ce temps l'homme de la Renaissance commence à comprendre l'existence des lois mécaniques qui assurent soit le mouvement des astres et de la terre soit les phénomènes de la vie extérieure de l'homme et des animaux soit les propriétés mécaniques de la matière

Contrairement à Copernic les philosophes antiaristotéliques préparent cette immense révolution de l'esprit de recherche Et une heureuse rencontre une rencontre qui aurait produit des effets extraordinaires a lieu en 1592 Galilée reçoit cette année la chaire de mathématiques à l'Université de Padoue par une coïncidence dont la signification est profonde le fondateur de la science expérimentale naît en 1564 l'année même de la mort de Vésale! Aussitôt une grande amitié unit Galilée à Fabricius d'Acquapendente qui devient son médecin et continuera à le soigner même quand Galilée passera à l'Université de Pise Or il est absolument certain que les deux maîtres de notre Athlénée échangèrent leur avis sur des problèmes d'anatomie des

organes des sens sur la physique des rayons de lumière ou de l'onde sonore. J'ai pu retrouver dans les œuvres de Galilée des pages dédiées à la physiologie des organes de sens : en 1622 il publia une synthèse des connaissances qu'il avait pu acquérir grâce à ses études et à ses échanges de vue avec les anatomistes de Padoue sur la physiologie de l'audition et de la vision. Il affirme dans son style classique et d'une extrême clarté que le phénomène physique qui est à la base des excitations provoquées par les agents extérieurs et les modifications qui se produisent soit dans les liquides contenues dans les bulbes oculaires soit dans les osselets de l'oreille moyenne étaient deux phénomènes tout à fait différents dans leur essence.

Nous ne trouvons aucun mot dans ces pages merveilleuses sur les phénomènes psychiques qui sont la conséquence des modifications des tissus sensoriels produites par les stimulations : c'est à dire sur le mécanisme de la sensation. Ce silence est le témoin de la prudence extrême du grand mathématicien devant des faits que la nouvelle physique ne pouvait pas encore expliquer. En même temps ce silence signifiait le refus des idées galiléennes sur la physiologie sensorielle qui appartenaient à l'enseignement officiel mais avaient été ébranlées à la suite des découvertes anatomiques déjà au moment de la présence de Galilée à l'Université de Padoue.

Là en effet la moderne histoire de la médecine est en train de démontrer par des recherches excessivement difficiles l'influence exercée par les physiciens du XVI^e siècle sur la physiologie des organes des sens. On sait que celle-ci ne devait s'affirmer d'une façon définitive qu'en plein XVII^e siècle avec les œuvres de Descartes de Duverney de Borelli et de Molinetti ce dernier aussi professeur à Padoue. Et l'on connaît bien aussi l'influence que Descartes reçut de Galilée à travers l'abbé Mersenne son élève à Padoue et qui fut l'intermédiaire entre les deux grands savants.

Les anatomistes padouans de la Renaissance avec une technique excessivement simple sans l'aide du microscope après avoir enlevé l'oreille externe et moyenne s'étaient jetés dans l'observation et la description minutieuse des cavités de l'oreille interne : quand on regarde les splendides gravures de leurs œuvres ou chaque dessin est posé sur la feuille blanche avec un esprit artistique et une extraordinaire harmonie des espaces blancs et noirs nous sommes saisis d'un sentiment d'étonnement et de profonde admiration. Le crinil cochléaire le vestibule les trois canaux semicirculaires sont reproduits avec une perfection absolue dépourvus de toute formation membraneuse (car dans le cadavre chaque tissu sauf l'osseux disparut bien vite) ces minuscules cavités étaient regardées mesurées comparées avec un travail patient et infatigable : si l'œil ne pouvait pas dominer la forme complète de la cavité on faisait un effort d'intégration géométrique pour pouvoir tracer un dessin exact de toute la cavité.

Les cavités labyrinthiques sont définies en faisant appel à des formations naturelles connues : le labyrinthe l'entonnoir les petits tunnels qui font plusieurs courbes. Et c'est Cressenius qui a décrit l'appareil vestibulaire du jasson pour la première fois (1461).

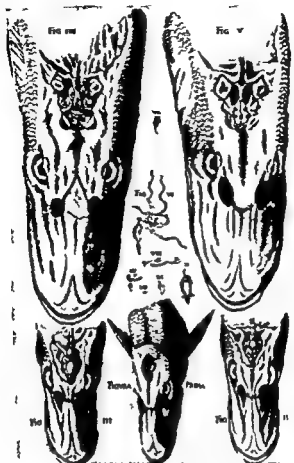


FIG. 1. L'appareil vestibulaire du poisson : première description faite par J. Casseri (1552-1616) lecteur d'anatomie à l'Université de Padoue (*Pentaesthesion. De quinque sensibus liber*, Venise, 1609).

Or, si on lit avec attention les thèses que ces précurseurs proposent pour expliquer à quoi ces cavités doivent servir, il nous apparaît avec une pleine évidence qu'ils avaient parfaitement compris que les ondes sonores, dès qu'elles sont arrivées de l'extérieur à ces cavités, y produisent de nouvelles ondes sonores qui présentent différents caractères selon la grandeur, la longueur, les courbes des cavités.

Fabrizius affirme même que la différence entre les sensations de sons graves et des sons aigus dépend de la forme des cavités de l'oreille interne : « Ex foraminibus alia ampliora sunt alia angustiora ita facta ut ad eam soni differentiam admittendum se accomodent quae per gravem et acutum variam » (« Parmi ces cavités, certaines sont plus larges, certaines plus étroites, de façon que les sons s'adaptent à cette différence, selon qu'ils sont aigus ou graves »).

Il prévoit donc, avec son esprit d'observation et son intuition fondée sur

La comparaison entre les données anatomiques et les lois physiques connues à ce temps là la théorie de Helmholtz dont les principes essentiels sont bien valables aujourd'hui. Et il est hors de doute que la description anatomique des cavités de l'oreille interne nous apparaît conduite et accomplie d'un point de vue strictement méthodologique et iconographique d'une façon bien supérieure à celle qui a été suivie par un Retzius ou un Bast.

Via Altinate 37 Padova (Italie)

KURZE ÜBERSICHT ÜBER DIE HISTORISCHE AUSSTELLUNG VON OTOLOGIEBÜCHERN IM ARCHIVIO ANTICO DER PADUA UNIVERSITÄT (29.8.1960)

Prof. LORIS PREMUDA

Direktor des Institutes der Geschichte der Medizin der Universität

Es ist nicht möglich in Padua zu sein ohne die Gelegenheit zu benutzen auch ein wenig vom ganz eigenartigen zauberlichen Duft zu atmen der sich von der Geschichte der Universitätseinrichtungen — und besonders von den Medizeinrichtungen — herausbreitet. Der sehr geschätzte Meister und Freund Prof. Michele Arslan, hat dem Medizinhistoriker eine günstige Möglichkeit geboten die Ausstellung alter Texte zu kommentieren die ich selbst vorbereitet habe und welche Erwerbe der Kenntnisse über das Ohr vom 15. bis zum 18. Jahrhundert betreffen. Es ist deshalb zu meiner grossen Freude und Ehre dass ich dem so ausgewählten Publikum des „Collegium Oto Rhino Laryngologicum Amicitiae Sacrum“ sprechen darf.

Der grosse Kliniker und Historiker Adam Politzer (1835–1920) ausserte sich in seiner „Geschichte der Ohrenheilkunde“ im Sinne dass Italien als die Wiege der Ohrenanatomie zu bezeichnen ist. Diese Bemerkung entspricht der Wahrheit. In der Wende eines einzelnen Jahrhunderts vom Ende des 15. zum Ende des 16. Jahrhunderts bereichern sich entscheidend die morphologischen Kenntnisse besonders des äusseren Ohres und des Mittelohrs aber auch die des inneren Ohres indem diese Kenntnisse an Stelle der bescheidenen galenischen Begriffe und der öfters spekulativen oder phantastischen Verfasser vom Mittelalter treten.

Der moderne Forscher stellt sich die Frage warum diese rasche Blüte gerade zu dieser Zeit zustande gekommen ist. Das ist die Zeit der wissenschaftlichen Renaissance, die durch die humanistische Bewegung eine Brücke zwischen dem alten und dem neuen Denken schlägt. Schon im 15. Jahrhundert hatten Kunst und Wissenschaft geheiratet um ein gemeinsames Ziel zu erreichen: Kenntnis der Natur. Im 16. Jahrhundert ist das Erwachen kraftvoll und gerade in Padua werden neue Fragen im Studium der Anatomie gestellt. Die Genialität der paduaner Forscher senkt ihre Wurzeln tief in die methodologische Erneuerung die vor allem auf einer aufmerksamen und genauen Beobachtung gegründet ist. Systematisch werden hier in Padua die neuen Methoden angebracht die sezierende die vivisezierende die beweisende (im anatomischen Theater) die vergleichende Methode.

Die schweren in folio die an der heutigen Ausstellung zum Vorschein gebracht werden sind alle seltene Ausgaben. In jeder auch wer kein Historiker ist kann sich ein Bild davon machen welches die ausschlaggebenden Etappen der jahrhundertlangen Forschungen über die Ohrenanatomie sind wenn er auch nur flüchtig diese in chronologischer Ordnung eingereihten Bände nachschlägt.

So wird in *De administracionibus anatomicis* von Galen (131–201) über die Topographie der Schädelknochen und besonders der Schädelnahte erörtert und ziemlich genau das siebente und achte Paar Gehirnnerven geschildert die der grosse Gelehrte des Altertums in ein einziges fünftes Paar einschliesst.

Interessant ist die Bemerkung von Berengario da Carpi (1460–1530), der „duo ossiculi“ kennt und bemerkt, wie für die Prüfung der anatomischen Hohlheit des Schädels gewisse Mittel nötig sind „ad haec bene videnda requiritur docta manus cum tenaculis, falce ferre et malleo apto“ Bei Achillini (1463–1512) spricht man deutlich von „miringa“, die später Falloppia beschreiben und „Tympanus“ nennen wird.

Vesali (1514–61), der Reformator der Anatomie, gibt uns deutliche Beschreibungen von „malleus“ und „incus“ (Hammer und Amboss), die — wie Alessandro Fioretti, Mitarbeiter von Arslan und mir, genau feststellen konnte — in der Zeitspanne zwischen Achillini und Berengario entdeckt wurden.

Ingrassia (1510–80), der auch für seine wichtigen Beiträge zur Gerichtsmedizin bekannt ist, berichtet uns von der in Neapel erfolgten Entdeckung des „tertium ossiculum“, „staphes“, das einem sizilianischen Steigbügel ähnlich ist.

Bartholomeo Eustachi (ca. 1500–1571), der in Rom seine Tätigkeit entfaltete, bildet eine der interessantesten Figuren in der Entwicklung der Kenntnisse über das Ohr. Ihm verdanken wir nicht nur die Entdeckung der „tuba pharyngo tympanica“, sondern auch des „modiolus“ und der Muskeln „tensor tympani“ und „stapedius“.

Gabriele Falloppia (1523–63), der grosse paduaner Meister, entdeckte und beschrieb die „chorda tympani“, die halbkreisförmigen Kanäle, den Vorhofskanal, die „fenestra ovalis et rotunda“ und verleiht den Namen, ausser dem Tympanus, auch dem Labyrinth. Einer seiner Schüler, der Holländer Volcher Couter (1531–76), liefert uns in den sieben Kapiteln des „De auditu instrumento“ (1572) eine gewissenhafte synthetische Rundschau der Ohrenmorphologie, während Mercuriale (1530–1606), der auch in Padua manche Jahre weilte, uns ein erstes Handbuch klinischer Ohrenkunde, „De oculorum et aurium affectionibus praelectiones“ (1584) hinterlassen hat. Den beiden grossen Anatomen des letzten Abschnittes des 16. Jahrhunderts, Girolamo Fabrici d'Acquapendente (1533–1619) und Giulio Casseri (ca. 1552–1616) verdanken wir beziehungsweise „De visione voce auditu“ (1600) und „De vocis auditusque organis“ (1600). Im ersteren dieser zwei Werke lässt die Plastizität des Helldunkels im Kupferstich unter anderem beide Fenster und das Promontorium zum Vorschein kommen, wobei uns die cochleae zur Vorstellung tritt. Im anderen Werke findet das Labyrinth eine genauere Beschreibung, während die Forschungen über die vergleichende Anatomie uns vollkommenere Angaben über den ganzen Ohrenapparat anbieten.

Weiterhin ist „De aure humana“ das Grundbuch Valsalvas (1666–1723), eines Schülers Malpighis, bemerkenswert und das gilt auch für Morgagnis (1682–1771) monumentales Werk.

Fast siebenzig Jahre lang lehrte Giovan Battista Morgagni zu Padua, ihm verdankt man die endgültige Einführung des anatomischen Denkens in die Pathologie. Von der positiven Objektivität, die Morgagni in seinen Forschungen anwandte, existiert ein Beweisstück, unter zahlreichen anderen, das die Ohrenkunde betrifft. Die klangvolle Entdeckung der „aquidotti“ seitens des damals jungen Anatomen Domenico Cotugno, liess den paduaner Meister veräutzt und voll Zweifel und wurde nur dann überzeugt, wenn sich der südlandische Student persönlich bei ihm in Padua meldete und ihm die Beweise seiner Entdeckung mit ergebener Ehrfurcht vorlegte. Als Humanist im weitesten Sinne des Wortes, lässt er uns die mächtige Vielseitigkeit seiner Bildung fühlen, wenn er in seinem Werke „De sedibus“ von den Entzündungsvorgängen des Schlund- und Kehlkopfs redet und, dem lateinischen Verfasser Macrobius folgend, Gelegenheit findet, die Opfer an die Göttin Angerona zu erwähnen, die das römische Volk durchführte, um sich von einer lästigen Beschwerde, der Mandelentzündung, zu befreien. In seinem Vorwort (1739) zu den eigenen Briefen über Valsalvas Schreiben,

die er selbst herausgab (1740–41) betont der paduaner Meister mit Scharfe folgendes, Ohr ist ein einziges Wort doch wieviele winzige Teile setzen es zusammen mit denen man sich einzeln und eingehend zu befassen hat! Dem Vermerk über die Schwierigkeit der Probleme der Ohrenkunde lasst er, in elf seiner achtzehn Briefe, eine recht echte Abhandlung übers Ohr — nach den Richtlinien von Valsalvas *De aere humana* — folgen. Den Vergleichen und den geschichtlichen und philosophischen Erörterungen fügen sich Schilderungen hinzu die auf der direkten Beobachtung und den von der vergleichenden Anatomie gelieferten Angaben gegründet sind.

Unsere Übersicht schliesst sich mit Cotugno's (1736–1822) Werk *De aquaeductibus auris humanae internae anatomica dissertatio* (1761) und mit Scarpa's Werk, *De structura fenestrae rotundae auris et de tympano secundario* (1772) der Ertere hat dem inneren Ohr eingehende und bedeutende Forschungen gewidmet und der Letztere kam durch vergleichender Anatomie Forschungen zum Schluss dass die *fenestra rotunda* eine grosse Rolle wie ein sekundärer Tympanus in der Gehorfunktion spielt.

Gegenüber der Geschichte von umfassenderen Problemen die einen höheren Widerhall in der Entwicklung der ärztlichen und wissenschaftlichen Errungenschaften im Allgemeinen finden ist das sich mit der Anatomie des Ohres befassende Problem relativ gering es weist doch Erscheinungen und Gründe auf die es höchst interessant machen. Die Geschichte der Ohrenheilkunde ist nicht damit geschlossen in ihrem Laufe sind Sie geschätzte Meister heute gewiss die würdigsten Fortsetzer und Vertreter!

BESUCH AM ANATOMISCHEN THEATER VON FABRICI D'ACQUAPENDENTE

Nun befinden wir uns in dem Gebäude wo seit über viereinhalb Jahrhunderten, unsere in 1222 entstandene Universität ihren Sitz hat. Hier sind wir nämlich im anatomischen Theater das nach Fabrici d'Acquapendente genannt ist. Es war in 1524 bereit und wurde am 16. Januar 1595 eingeweiht. Diese Errungenschaft stellt — auch als Denkmal betrachtet — den Schluss eines Jahrhunderts anatomischer Forschungen dar die für das Schicksal der modernen Medizin äusserst entscheidend waren. Das Theater das nach dem Entwurf von Fra Paolo Sarpi gebaut wurde war dem Publikum von Gelehrten und Studenten ohne Einschränkungen offen wobei man sich nach dem Motto richtete, *Universa Universitas Patavina Libertas*. Von 1515 bis 1872 wurde hier gelehrt und der menschliche Körper sezziert.

Der Bau sieht aus wie ein umgewandter Kegelmantel auf elliptischem Grund mit sechs hölzernen Stufen die mit einem aus fein geschnittenen kleinen Pfeilern gebildeten Geländer versehen sind. Man kann den Platz der für den beweglichen Tisch für den Leichnam reserviert war und den Stuhl für den Professor unterscheiden. Das Theater wurde drei Jahrhunderte lang mit Fackeln und Kerzen beleuchtet. Erst 1844 bekam es Tageslicht dadurch dass vier Seitenfenster erweitert wurden.

Hier lehrten Fabrici d'Acquapendente, Casseri, van Spiegel, hier wies Morgagni dem modernen ärztlichen Denken neue anatomische Richtlinien auf und hier errichtete ihm die *Natio Germanica* ein Marmorbild. In 1769 das noch heute zu sehen ist. Es ist das einzige Theater auf der Welt das voll Altertums noch heute unversehrt aufbewahrt ist. Dieses Theater ist der Leertempel der modernen Anatomie und folglich des modernen ärztlichen Denkens.

DIAGNOSTIC DES VERTIGES

Film sonore en couleur

A. MONTAUDON

Geneve, Suisse

Clinique O R L Hôpital Cantonal

ELECTROENCEPHALOGRAPHIC AUDIOMETRY

Film

G. FERRENI and L. FIORI RATTI

Rome Italy

The authors present a new method for electroencephalographic audiometry, carried out in a sound proof room. The new evaluation of electrographic results permits not only a re evaluation of electroencephalographic audiometry for audiometric purposes but contributes also to the topographic diagnosis of the lesion.

A. P. a. Priscilla Roma

INTRA VENULE PHENOMENA

A still and cine photography demonstration and discussion

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From the Department of Otolaryngology College of Physicians and Surgeons under grants from the Philip Hanson Hiss Jr. Memorial Fund, The Abbott Laboratories and The Merck Institute for Therapeutic Research

Still and cine photography of the events which take place in and around venules of 50 micra or less diameter with trauma, hypersensitivity and infection. The importance of these events in the development of symptoms and pathology as well as methods of control will be discussed.

In every tissue of the body the blood vessels are involved with physiologic as well as pathologic changes. This has been known since the time of Cohnheim and perhaps before. To be considered are arterioles to supply the capillaries and venules to drain them, not to mention arteriovenous and other types of shunts and the lymphatics. However, the most numerous of all the active vessels are venules, for more blood passes through the venules than capillaries in normal tissue because here many of the capillaries are inactive.

Now after nearly twenty years of study of microvascular reactions to threats be they traumatic, infectious, toxic, immunologic, neurologic or psychosomatic, it has become apparent that the phenomena that occur within venules and their walls especially at junctions are of the greatest importance. Let us hasten to say that this has become a growing conviction among many other workers in the field of microcirculation notably Bloch, Ebert, Fulton, Irwin, the Knisely's, Lutz and Weifach to mention but a few.

Previously the emphasis has been on the vasomotor effects which produce microcirculatory changes in arterioles, venules, and shunts. These secondarily effect capillary blood flow but are often transient and minor compared to what goes on inside the small venules. Fig. 1. Such vasomotor effects and some of the pathology therefrom were reported before this society by the author in 1952 and still seem valid. But improved techniques and longer observations of the tissue however have demonstrated more clearly the other modus operandi of the microcirculation under various stresses is more primitive and at the local cellular level and even to a restudy of the movements and habits of single cell especially leukocytes (Fig. 2) and mast cells (Fig. 3).

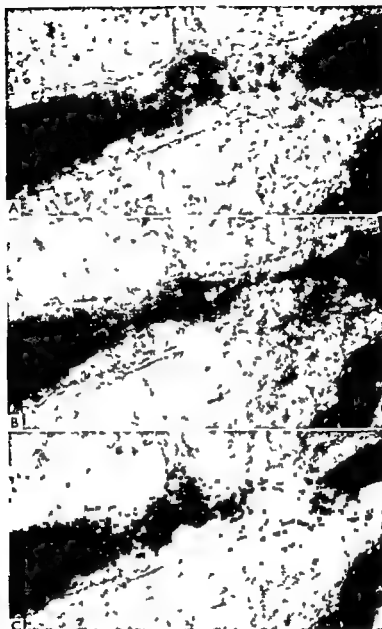


FIG. 1. Development of thrombotic emboli in living hamster cheek pouch venule measuring 50 microns. White cells and platelets stick to the walls and compromise the flow.

A. Slow beginning of build up. B. Partial occlusion. C. Complete occlusion which may or may not persist. If the thrombus breaks off and enters the blood stream it may produce symptoms in other parts of the body (see Fig. 5).

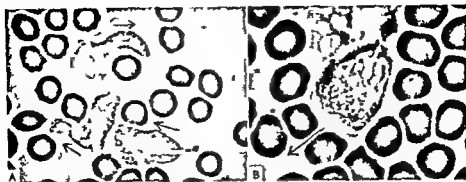


FIG. 2 Phase contrast cinematography of a drop of fresh human blood under a cover slip. Arrows indicate direction of motion. Erythrocytes indicate scale.

A: Single frame from 16 mm film indicating active motion of leucocytes. B: Single frame at higher magnification showing polymorphonuclear neutrophils trailing some debris and four platelets (upper center).

This has led to an increased study of the intravascular venule phenomena which are the subject of the present paper.

The experimental evidence for symptomatology is less clear cut but one sees venule engorgement and intermittent sluggish flow of blood containing red and white cell lumps in the conjunctival venules of patients with Meniere's disease (Fig. 4). The author has shown that tinnitus can often be clearly stopped or altered when changing such blood flow by the use of intravenous procaine which increases the stroke volume of the heart and thus speeds up circulation. Yet Perlman and Kimura report few arterioles in the stria vascularis and have shown pretty conclusively that there are no changes in the diameter of the stria vessels with sympathomimetic drugs. Thus the theory that direct vessel wall muscle constriction is a cause of the symptomatology of Meniere's disease such as has been indicated by Hilger and others is probably incorrect. At the same time Perlman and Kimura, Irwin and observers in our own laboratory have seen white thrombi (Fig. 5) in the circulation of the inner ears of guinea pigs. These white thrombi sometimes block the circulation in the inner ear so that there is no question that the more primitive reactions to tissue injury or stress mentioned above occur in the inner ear at least in guinea pigs and must occasionally cause symptoms. Such intermittent venule blockage would explain the intermittency of Meniere's disease attacks. The experiments mentioned above with the improvement of tinnitus after intravenous procaine therapy coincidental to the increased flow and picking up of sludge patterns also points to the lumpy blood in the small blood vessels of the inner ear as a probable causative factor for inner ear symptoms in human beings.

With this background in mind it seemed to me wise to demonstrate in living tissue these almost universal intravascular venule phenomena to ear, nose and throat clinicians. The demonstration should help to explain the basis for using therapy directed toward changing intravascular phenomena



FIG 6 Thrombo-emboli (T.E.) developing in a venule measuring circa 50 micra in vivo Hamster cheek pouch in phase contrast. Note change in size of occluding mass as red cells flow past in the direction indicated by the arrows Note smaller venules also and arteriole at the left.

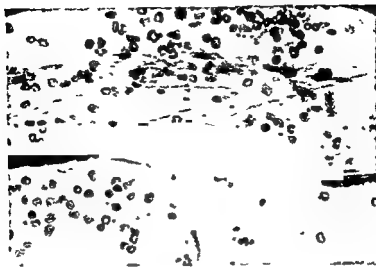


FIG 7 Hemolyzed erythrocytes thrombosis in dilated region of a rabbit mesentery venule This hemolysis when it occurs takes place without apparent cause in a few seconds (Zenker fixation, Giemsa stain, red cells indicate scale)

body When these microthrombi develop in the lung for example they are most likely to throw off emboli to distant organs

"White spaces" indicating microscopic thrombo emboli can be seen in patients with systemic disease Bloch estimates that 24 to 98 hours after an acute myocardial infarct, for example, that red aggregates from the morbid area in the heart increase in number and rigidity so that they intermittently block the arterioles in the general circulation for a few to 30 seconds or more and that these aggregates involve from 50% to 25% of the circulating erythrocytes Red aggregates and "white thrombi" can be seen in the conjunctival vessels of patients with almost any disease but are particularly prominent when the *in vitro* erythrocyte sedimentation rate is high The inner ear should be particularly vulnerable to circulating emboli It is unique because its blood supply is terminal Furthermore, because it is so sensitive, minute changes in its blood supply should produce dramatic and measurable reactions Cf Fowler and Fowler

Clearing out of the microscopic thrombi and improvement of the general circulation by such agents as nicotinic acid, anti coagulants, and intravenous procaine seems a desirable goal for therapeutics Our experiments are beginning to indicate anti inflammatory effects with anticoagulants and antiserotonins Nicotinic acid has been used for Meniere's disease and we now use it for Bell's Palsy, Sudden Deafness, Difficult Swallowing (globus hystericus) and Nuchal Occipital Headaches How much is placebo effect, especially in the last two named is difficult to say, but the results are often very satisfactory and certainly the hypothesis that improved circulation should be helpful to reduce inflammatory and ischemic reactions of all kinds, is attractive It is not inconceivable that even a placebo or strong reassurance, as administered by a

REACTION OF NASAL MUCOSA IN RABBITS AFTER CERVICAL SYMPATHECTOMY

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PROBLEM

In examining the reactions of the nasal mucosa in patients with vasomotor rhinitis we have found that these reactions are more pronounced than in normal individuals (Table 1). Thus we have been able to come to the conclusion that such a mucosa may in its reaction show a classical picture of vasomotor rhinitis even from the physiological irritation to which a normal mucosa does not react. Furthermore, in such cases both exogenic and endogenic irritation from any place in the organism may be reflected upon the hypersensitive nasal mucosa. Only thus we can explain the physical allergy in which not only the irritation of the nasal mucosa but also that of the outer skin or even of the oral cavity can lead to exaggerated reactions of the nasal mucosa.

After this we tried to discover the reason for such exaggerated reactions. Having investigated cases of vasomotor rhinitis over a period of ten years we came to the conclusion that disturbances of the vegetative function of the nasal mucosa for widely differing reasons are the cause of such reactions (Table 2). We therefore artificially severed the sympathetic net of the nasal mucosa in order to detect how it would react to mechanical, physical and chemical irritations. Of course, this is the maximal application of these vegetative unbalances, but it is only in this way that we can detect such divergent reactions. Moreover, this experimental work done on rabbits cannot be quite adequately applied to man. We are of the opinion that these unbalances are still more pronounced in a human being because the central nervous system

TABLE 1. *The average value of the difference of the blood pressure, pulse and leucocyte before and after cocaine anasthesia of the nasal mucosa*

	Number of cases	Blood pressure mm Hg	Pulse	Leucocyte
Vasomotor rhinitis	22	20-35	10-25	600-2000
State after laryngectomy	8	5-15	5-10	200-400
Ozaena	6	5-10	8-14	300-500
Mentire sedation	6	10-20	10-15	400-900
Without any affection	10	5-10	5-10	100-500

TABLE 1. Vasomotor rhinitis

```

(a) Allergic      Seasonal
                  Perennial
(b) Non allergic  Endocrine
                  Physical
                  Toxic (post infectious)
                  Psychosomatic
                  Postoperative
                  ↓
Vegetative disturbances either general or local in the nose
                  ↓
Vasomotor and gland secretion troubles
                  ↓
Typical attacks of vasomotor rhinitis

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having reached the stage of development it has in man, can even more strongly influence the appearance of such exaggerated reactions

EXPERIMENTS

We have done our experimental work on chinchilla rabbits weighing between 3000 and 4000 g and between the ages of six months and one year. The sympathetic control of the head in rabbits is performed by the upper cervical ganglion (Fig. 1). In order to obtain sympathetic denervation, in addition to removing the ganglion, we, as a rule, denervated the carotid. After that we connected the femoral artery on the left or right side with the lymo-



Fig. 1

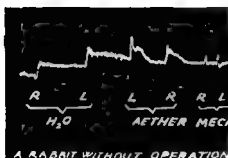


FIG 2

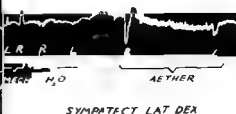


FIG 3

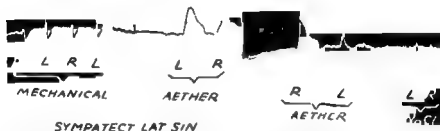


FIG 4



FIG 5

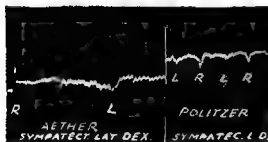


FIG 6



FIG 7

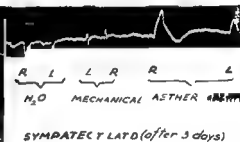


FIG 8

graph and registered the pressure. Then we applied the mechanical, physical or chemical irritation to the nasal mucosa the mechanical by means of a small roll of cotton wool or of a round, smooth catheter, the physical by means of cold water, and the chemical by means of ether (5 to 10 drops). The result of such irritations was a greater or lesser blood pressure in the rabbits. All the rabbits were narcotised with urethane so that all of them were quite quiet all through the experiment, which was done in order to avoid certain defense reactions which might have influenced the kymographic curve. The experiment was successful in seven cases out of ten. In five cases sympathetic denervation of the neck was made, in one case the reflexes were examined without the exclusion of the sympathetic nerve, and in another they were examined on the third day after the cervical sympathectomy (Figs 2-8).

COMMENTARY

In 1955 Goodman & Gilman stated that vegetative denervation causes a cell to become more sensitive to chemical mediators and to other non specific irritations. There exist various opinions as to the cause of the increased sensitivity after the denervation of the cell but the greatest importance is attributed to the disturbed or the increased permeability of the cell. Every irritation upon a denerved cell or a denerved organ causes functional changes and it is more difficult for them to return to the normal level as there is no regulatory mechanism that can paralyse the influence of other factors and lead to equilibrium.

Of course the stronger or the more different the irritations the stronger will be the reactive changes of the mucosa. We endeavoured to make the mechanical irritation of the mucosa as small as possible and to prevent the traumatization of the mucosa. To such mechanical irritation the reaction response on the kymographic curve was not so pronounced as in the case of physical irritations with cold water and most pronounced in the case of chemical irritation with ether. By this experiment we have also confirmed that the nasal mucosa has wide reflexive connections with all the organism. This means that many irritations in the organism can act through these reflexes of the mechanism upon the nasal mucosa especially in cases where there exist vegetative irregularities. This vegetative unbalance can be general or localized upon the mucosa as a result of previous infections or the trauma of the nose itself. Every irritation on such a mucosa leads in greater or lesser degree to the disequilibrium between the cells and the intercellular space. This will make possible a greater formation of various intermediate metabolites or in the case of the removal of the sympathetic nerve increased functioning of H substance. These matters secondarily lead to changes in blood vessels, nerves and the glands of mucosa and the final result is its functional disturbance. The relation between the sympathetic nerve and the parasympathetic nerve on the nasal mucosa can vary greatly. Our experimental work has come down to one variant of denervation of a system. However many factors that change this equilibrium can lead to the widely differing relation between these two systems so that the picture of the nasal mucosa will also be different. This is in fact the reason why in vasomotor rhinitis we can see many different relations in clinical symptomatology.

CONCLUSION

The authors have studied the reflexory reactions of the nasal mucosa to mechanical, physical and chemical irritation after the cervical sympathectomy in seven rabbits. They have registered these reactions through the femoral artery upon the kymographic curve. They have found these reactions are much more pronounced on the side of the nasal mucosa which has been denervated.

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Buconjiceva 14, Zagreb

DISCUSSION

C-4 Hamberger Dr Krajina has made a very good contribution to an important problem. Ten years ago I had some opportunities of examining patients who had been operated upon at the neurosurgical clinic of Serafimerlasarettet in Stockholm for angina pectoris. The operation consisted of the removal of the stellate ganglion, and section of the second and third, or fourth, thoracic ganglia.

Among these patients the operation was frequently followed by signs and symptoms from the nose. Out of 68 patients who were observed for a sufficiently long time 31 complained of stuffiness in the nose, which usually developed within one to three months. In 19 patients the complaint was of little significance, requiring no treatment, but in 16 patients it became a major complaint, sometimes interfering with sleep, making local application of ephedrine or even surgical treatment necessary.

In some patients roentgenograms showed an opacity of the paranasal sinuses without any clinical symptoms of sinusitis. The patient had chronic atrophic rhinitis for some years before operation. After left-sided operation the patient developed nasal congestion, with hyperplasia of the mucous membranes. In most of the cases a rhinoscopic examination showed considerable hyperemia and hyperplasia on the operated side, specially on the nasal septum. The complaints were more marked after a bilateral operation than after a unilateral. We have seen the same changes in the mucous membrane of the nose in man that Dr Krajina has shown in rabbits.

V. Hlaváček Besonders interessante Versuche haben eine grosse Bedeutung auch für den Mechanismus der allergischen Reaktionen. Ich habe einen Kranken nach Stellektomie gesehen, wo sich an der operierten Seite vasomotorische Schnupfen entwickelte und Fowler, Jun. hatte in einem ähnlichen Falle sogar eine Hypereosinophilie festgestellt. Daraus geht hervor, dass eine Imbalanz des vegetativen Systems entscheidende Rolle spielt, denn man durch Resektion des Sympathikus auch Schwarzmänn'schen Phänomen beeinflussen kann. Das gestörte Gleichgewicht dieses Apparates haben wir auch bei unseren Kranken mit allergischen, vasomotorischen Schnupfen nachgewiesen. Es scheint, dass diese gestörte Gleichgewichtigkeit des vegetativen Nervensystems in Folge der Sensibilisation des Organismus entstehen kann, wie es einige Autoren wie Ado, Hein und andere voraussetzen.

H. A. E. van Dishoeck In our attempts to discriminate between allergic and non-specific hypersensitivity in my clinic van Lier studied the excitability of the nose by means of different pepper solutions. Normally a short reaction consisting of swelling, secretion and occasionally sneezing results.

However, we found that in patients suffering from Ménière's disease, in whom ganglion stellatum anaesthesia was done, that on the side of this treatment, the nose presented a much stronger reaction. Indeed this reaction was similar to an attack of rhinitis vasomotoria.

Thus blockade of the sympathetic nerve brings the patient into a condition of latent vasomotor rhinitis and an attack may follow every relatively small excitation.

P. Ardouin. Il est essentiel de distinguer l'action de la sympathectomie sur chacune des deux zones des fosses nasales.

1° D'une part, sur la zone respiratoire, dominée par la présence du maxillo turbinale (cornet inférieur très vascularisé), et à laquelle vient s'ajouter, chez le lapin, l'ensemble naso sinusien constitué par le sinus maxillaire doublé de la cavité du naso turbinale. Toutes ces muqueuses réagissent violemment à la moindre modification circulatoire.

2° D'autre part, sur la zone ethmoïdo olfactive, dont les réactions sont d'ordre neuro sensoriel, car elles conduisent directement vers le diencéphale par le moyen des cellules de Schultze, de la voie olfactive et de l'hippocampe.

L'ÉVOLUTION DES CAVITÉS PARANASALES DE L'HOMME

Étude phylétique et embryologique

P. ARDOUIN
Tours France

L'auteur étudie le développement des cavités paranasales chez les vertébrés en particulier chez les mammifères les grands primates et chez l'homme

Il fait ensuite une étude embryologique des cavités sinusiennes de l'homme et il montre comment la station verticale de l'homo sapiens a conduit au développement spécial de l'ethmoïde et du complexe ethmoïdo-frontal humain

La réalisation anatomique des cavités paranasales de l'Homme est intimement liée d'une part à l'évolution de la fonction olfactive et d'autre part aux dispositions successivement adoptées par le squelette céphalique des vertébrés. Nous précisons toutefois que cette étude n'intéresse pas l'organe de l'olfaction envisagé du point de vue fonctionnel mais uniquement le développement des cavités aériennes annexées aux fosses nasales et creusées dans les formations osseuses de la face et du crâne

Des quatre cavités sinusiennes le *sinus maxillaire* est le plus ancien du double point de vue de la phylogénie et de l'embryologie. C'est lui qui apparaît le premier dans la série animale de même que sur l'embryon humain de 40 mm c'est par une encoche inférieure du mur moyen embryonnaire que se dessine la première diverticule des fosses nasales dirigée vers le mésenchyme du maxillaire supérieur

Le *sinus frontal* qui se manifeste à partir des Mammifères n'est cependant pas toujours constant dans tous les groupes. Lorsqu'il existe il se présente souvent comme une sorte de prolongement supérieur du sinus maxillaire qu'il coiffe à la manière d'une calotte. Parfois même chez les Primates et chez certains Anthropoïdes comme chez l'orang, le sinus maxillaire a une tendance à envahir l'os frontal et le sphénoïde dont les cavités respectives communiquent alors directement et largement avec l'autre d'Highmore

De telle sorte que jusqu'à l'Homme c'est le sinus maxillaire qui semble constituer la cavité paranasale centrale autour de laquelle gravitent les sinus frontaux et sphénoïdaux les cellules ethmoïdales étant pratiquement inexistantes

La position de l'*Ethmoïde* du point de vue évolutif est tout à fait spéciale. On pourrait même ajouter que son importance dépasse le cadre étroit des fosses nasales et que la connaissance exacte de son apparition et de son développement est primordiale pour la compréhension de l'Homme lui-même

C'est qu'en effet le labyrinthe ethmoïdal tel qu'il est décrit dans les traités d'anatomie humaine est une acquisition *spécifiquement humaine*

Aucun Mammifère aucun Primate pas même le chimpanzé ne possède de loin un appareil ethmoïdal comparable à celui de l'Homme. C'est ici qu'interviennent des modifications de stature et d'attitude qui vont bouleverser l'agencement des cavités paranasales au bénéfice de l'ethmoïde qui va désormais déposséder le sinus maxillaire de sa primauté.

Tout s'explique en premier lieu par l'embryologie.

L'infundibulum ethmoïdal embryonnaire de l'Homme colonisera tout d'abord le maxillaire supérieur pour respecter la règle immuable de la phylogenèse mais trouvant devant elle un *squelette cephalique nouvellement adapté à la pneumatisation* la gouttière ethmoïdale n'hésitera pas à devenir envahissante et creusera de nouvelles cavités dans les os du voisinage de telle sorte qu'elle *incorporera finalement dans son propre système* les sinus ancestraux c'est à dire le sinus frontal et le sinus ethmoïdal et que le sinus maxillaire se trouvera lui aussi soumis à l'emprise de l'ethmoïde.

C'est ainsi qu'elle réalisera

- en bas la cellule ethmoïdo maxillaire c'est à dire le sinus maxillaire
- en avant et en bas les cellules ethmoïdo unciformiennes
- en avant et en haut les cellules ethmoïdo aggeriennes et ethmoïdo unguales
- en haut la cellule ethmoïdo frontale c'est à dire le sinus frontal
- en arrière les cellules ethmoïdo bullaires
- en arrière et en haut les cellules ethmoïdo turbinales moyennes

Toutefois avant d'envahir le squelette voisin la première manifestation active de l'infundibulum embryonnaire restera toujours la pneumatisation du maxillaire supérieur et c'est ainsi que la première image des cavités paranasales de l'Homme sera représentée par une gouttière cephalique limitée en bas par la poche de l'utante du sinus maxillaire et en haut par le recessus fronto ethmoïdal.

Cette disposition initiale est essentielle car elle a pour résultat de promouvoir le sinus frontal *embryonnaire* au rang d'une coupole coiffant directement le sinus maxillaire *au dessus d'un canal maxillo frontal*.

Nous retrouvons là dans les premiers stades de l'ontogenèse la rectitude et la simplicité originelle de la lesion fronto maxillaire semblable à celle que l'on constatera à la fois chez les Mammifères macrosomatiques chez les Primates inférieurs et chez les grands Anthropoïdes.

Seul l'envahissement ethmoïdal tel qu'on l'observe chez l'homme viendra transformer l'aspect architectural de toutes les cavités pneumatiques de la face.

Il est de même que dans le groupe des Mammifères en y comprenant les Primates les cavités paranasales vivent sous un régime sinusal dominé par le sinus maxillaire de même on peut affirmer que chez l'Homme il s'agit d'un gouvernement ethmoïdal.

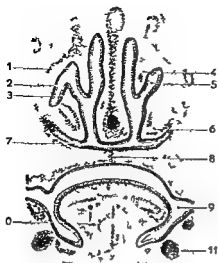


FIG 1



FIG 2

FIG 1 Coupe frontale de la partie antérieure de l'extrémité céphalique d'un embryon humain de 64 mm 1) récessus frontal — 2) infundibulum — 3) apophyse unciforme — 4) cornet moyen — 5) méat moyen — 6) cornet inférieur — 7) méat inférieur — 8) suture palatine — 9) cavité buccale — 10) langue — 11) cartilage de Meckel

FIG 2 Paroi externe des fosses nasales d'un embryon humain de 12 semaines. Notez la nette différenciation du méat moyen: apparition du début de la pointe du sinus maxillaire et du récessus frontal 1) sinus maxillaire — 2) récessus frontal — 3) infundibulum — 4) cornet moyen — 5) cornet supérieur — 6) méat supérieur — 7) cornet inférieur — 8) méat inférieur

Dans le premier cas les cavités sinusiennes paraissent avoir été conquises pour ainsi dire passivement et avoir été groupées autour d'une cavité unique creusée dans le maxillaire supérieur des vertèbres.

Dans le second cas au contraire il semble que l'on se trouve en présence d'une réalisation active dynamique (comme toutes les créations) provoquée ici par l'apparition d'une lame épithéliale de potentialité nouvelle: la lame ethmoïdale qui va conquérir de haute lutte la région frontale le sphénoïde et le maxillaire supérieur devenus ainsi tous les trois satellites de la nouvelle étoile sinusienne.

Quant au Sinus sphénoïdal il n'apparaît pas chez tous les Mammifères d'une façon constante.

Son sort est lié comme l'ont bien montré Dursy puis Van Gisle au développement de l'arrière cavité de la capsule nasale embryonnaire destinée à réaliser la chambre olfactive des Mammifères macrosomatiques.

Chez certains de ces animaux comme chez le chien le développement de la chambre olfactive s'arrête à ce stade et il n'existe pas de sinus sphénoïdal.

Chez d'autres comme chez le chat la constitution de la chambre olfactive n'est que le début de la réalisation sinusienne puis dans un second temps la capsule nasale postérieure pénètre dans le sphénoïde pour le pneumatiser.

Chez l'Homme le sinus sphénoïdal de l'adulte se construit également en deux étapes.

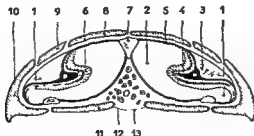


FIG. II — Coupe transversale des cavités olfactives et paranasales d'un Amphibien (*Plethodon glutinosus*) 1) sinus maxillaire — 2) cavité nasale principale — 3) portion cartilagineuse — 4) portion hyaline du cornet — 5) muqueuse olfactive — 6) épithélium olfactif tapissant la paroi latérale des fosses nasales et du sinus maxillaire — 7) cloison des fosses nasales — 8) os frontal — 9) os pré-frontal — 10) maxillaire supérieur — 11) palatin — 12) glande inter-maxillaire — 13) muqueuse buccale (d'après Widersheim)

1° Une première étape embryonnaire et postnatale qui correspond du point de vue phylogénétique à la chambre olfactive des Mammifères c'est le *paleo sinus*

2° Une deuxième étape infantile prépubertaire et pubertaire coïncidant avec la soudure ethmoïdo-sphénoïdale par le moyen des *osselets de Berlin* qui subissent à leur tour l'assaut pneumatissant de la muqueuse capsulo-nasale postérieure réalisant ainsi le sinus sphénoïdal définitif de l'adulte c'est le *neo sinus*

L'intérêt de ces diverses observations paraît évident

En effet ces réalisations successives de la phylogénèse puis de l'embryologie humaine vont nous expliquer pourquoi tout en adoptant un *usage ethmoïdal* nous eussions les cavités sinusiennes de l'Homme conserveront parfois le souvenir de leur disposition première et c'est ainsi qu'il ne si pas rare de voir chez l'Homme en dehors des relations fronto-ethmoïdo-nasales classiquement humaines un sinus frontal communiquant directement comme chez nos ancêtres avec le sinus maxillaire

Déjà signalé pour la première fois par Schaeffer en 1910 ce canal maxillo-frontal a été décrit de nouveau par Villar Fiol de Valence en 1928 et par le Professeur Terracot et nous même devant la Société Française de Laryngologie en 1956

Nous avons eu l'occasion de préciser que chez l'Homme cette communication fronto-maxillaire passait par la cellule de l'aggrégat nasal qui est l'homologue de l'ancien cornet naso-turbinal des Mammifères

Le second fait mis en lumière par la confrontation de ces deux disciplines est représenté par les cas assez rares chez l'Homme d'*agenésie du sinus sphénoïdal*. Dans ces conditions seul le *paleo sinus* persiste le développement embryonnaire s'étant arrêté au stade de la chambre olfactive des Mammifères

Nos recherches d'anatomie comparée ont porté sur quelques Mammifères d'une part et d'autre part sur certains Primates



FIG. 4. Paroi externe des fosses nasales du lapin. Remarquer 1) en avant le maxillo-turbinal 2) en arrière les ethmoïdo-turbinaux 3) au dessus des ethmoïdo-turbinaux on voit le naso-turbinal ouvert il recouvre le sinus maxillaire et se continuant en arrière vers l'os frontal.

Signalons cependant que la première manifestation d'une cavité annexe des fosses nasales apparaît pour la première fois chez les anoures sous la forme d'un diverticule infero-externe de la cavité principale tapissée comme elle d'un épithélium olfactif. C'est déjà le sinus maxillaire.

Les fosses nasales des Reptiles sont caractérisées par la présence sur leur paroi externe d'un cornet unique qui subdivise le cavum principal en deux parties : une partie supérieure strictement olfactive et une partie inférieure diverticulaire qui fait encore office de sinus maxillaire mais qui est cette fois tapissée d'un épithélium respiratoire.

Cette distinction en deux étages fonctionnels différents va se maintenir de nos jours dans toute la série jusqu'à l'Homme.

À partir des Mammifères nous voyons apparaître de véritables cavités aériennes annexes aux fosses nasales et ayant une tendance à pneumatiser non seulement le maxillaire supérieur mais aussi la région frontale et le sphénoïde.

Cependant parmi ces trois sinus seul le sinus maxillaire sera constant. Le sinus frontal et le sinus sphénoïdal seront en effet très souvent défaut. Quant aux cellules ethmoïdales elles seront ou bien absentes ou bien très rudimentaires jusqu'à l'Homme (à l'exception du chimpanzé).

D'autre part la paroi externe des fosses nasales des Mammifères possède toujours trois sortes de cornets :

1° Un cornet maxillo-turbinal situé sur un plan inférieur et faisant corps avec le maxillaire supérieur : c'est le futur cornet inférieur de l'Homme.

2° Une série de cornets ethmoïdaux toujours très régulièrement déroulés et dont le nombre va de trois à cinq suivant qu'il s'agit de Mammifères microsmatiques ou macrosmatiques.

3° Enfin un cornet que ne possède pas l'Homme qui est situé immédiatement au-dessus de l'os nasal : cornet très allongé dans le sens antéro-postérieur creusé d'une cavité et qui coiffe le maxillo-turbinal et les cornets ethmoïdaux : c'est l'os-turbinal.



Fig 10 ■ Paroi externe des fosses nasales du mouton. On voit également comme chez le lapin le maxillo-turbinal, les ethmoïdo-turbinaux et la cavité du naso-turbinal largement ouverte et qui communique en arrière avec le sinus frontal.



Fig 11 ■ Paroi externe des fosses nasales d'un Propithecus. Remarquez le drain placé directement dans le sinus maxillaire dont la cavité communique à plain canal avec le sinus frontal.

Tous les Mammifères possèdent un cornet naso-turbinal mais chez les Primates ce cornet perd de ses dimensions surtout chez les grands Primates ou il s'atrophie chez le gorille et chez l'orang.

Chez le chimpanzé qui est le plus proche de l'Homme le naso-turbinal n'est plus marqué que par une saillie qui se rapproche davantage du monticule de l'aggrégat humain que du naso-turbinal des Mammifères.

Parmi les Mammifères usuels nous avons étudié le lapin, le chevreau et le mouton. L'examen des cavités paranasales de ces animaux est riche en enseignement.

Chez le lapin seul existe le sinus maxillaire mais en l'absence de sinus



FIG. 8. Paroi externe des fosses nasales du chimpanzé (crâ. « sec »). On voit le drainage du sinus sphénoïdal dans le recessus éthmoïdal supérieur. Le sinus frontal se fraie directement dans une cellule éthmoïdale antérieure.



FIG. 9. Coupe sagittale d'une tête de chimpanzé revêtue de ses parties molles. La paroi externe des fosses nasales a une morphologie presque humaine.

Comment l'ensemble sinusien maxillo fronto sphénoïdal des Primates a-t-il été dépouillé de son unité au profit de l'éthmoïde ?

C'est ici que nous devons essayer de découvrir un coin du voile qui plane sur l'origine même de l'Homme.

On a écrit que le cerveau de l'Homme était comme *une fleur épanouie au sommet de sa tige vertébrale* et certains savants ont aussitôt froncé le sourcil car on admet difficilement que la science flirt avec la poésie ! Et cependant cette image poétique est bien l'expression d'une vérité scientifique.

Ce qui caractérise essentiellement l'Homme en effet c'est l'ampleur de son cerveau et bien que l'Homme soit très peu spécialisé du point de vue



FIG. 5 Paroi externe des fosses nasales du mouton. On voit également comme chez le lapin le conchilo turbinale les ethmoïdo turbinaires et la cavité du naso-turbinal largement ouverte et qui communique en arrière avec le sinus frontal



FIG. 6 Paroi externe des fosses nasales d'un Propithecus. Remarquez le drain placé directement dans le sinus maxillaire dont la cavité communique à plain canal avec le sinus frontal

Tous les Mammifères possèdent un cornet naso turbinale mais chez les Primates ce cornet perd de ses dimensions surtout chez les grands Primates ou il s'atrophie chez le gorille et chez l'orang

Chez le chimpanzé qui est le plus proche de l'Homme le naso turbinale n'est plus marqué que par une saillie qui se rapproche davantage du monticule de l'aggrégat humain que du naso turbinale des Mammifères

Parmi les Mammifères usuels nous avons étudié le lapin le chevreau et le mouton. L'examen des cavités paranasales de ces animaux est riche en enseignement

Chez le lapin seul existe le sinus maxillaire mais en l'absence de sinus



FIG 11 Paroi externe des fosses nasales du chimpanzé (crâne sec) On voit le drainage du sinus sphénoïdal dans le recessus ethmoïdo sphénoïdal Le sinus frontal se draine directement dans une cellule ethmoïdale antérieure

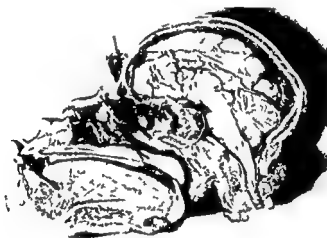


FIG 12 Coupe sagittale d'une tête de chimpanzé revêtue de ses parties molles La paroi externe des fosses nasales a une morphologie presque humaine

Comment l'ensemble sinusien maxillo fronto sphénoïdal des Primates a-t-il été dépoussé de son unité au profit de l'ethmoïde ?

C'est ici que nous devons essayer de découvrir un coin du voile qui plane sur l'origine même de l'Homme

On a écrit que le cerveau de l'Homme était comme *une fleur épanouie au sommet de sa tige vertébrale* et certains savants ont aussitôt froncé le sourcil car on admet difficilement que la science flirte avec la poésie ! Et cependant cette image poétique est bien l'expression d'une vérité scientifique

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somatique bien que sa morphologie générale le rapproche toujours des plus primitifs des Primates il existe cependant une exception à cette règle et cette exception est d'importance puisque la seule spécialisation choisie par l'Homme c'est la *spécialisation cérébrale*.

Chez l'Homme le néopallium le cortex cérébral a pris un développement extraordinaire il s'est effectivement épanoui au sens propre du terme étalant sa masse en recouvrant le cerveau olfactif le cerveau intermédiaire et même le cervelet.

Or cette tendance dominante de l'encéphale est ébauchée dans l'embryon de tous les Mammifères de même que sur l'embryon de tous les Mammifères et de tous les Primates en particulier se trouve représentée l'image d'un infundibulum muqueux de nature ethmoïdale.

Pourquoi donc l'Homme est-il le seul à avoir bénéficié de ces dispositions sinon grâce à une évolution particulière de sa boîte crânienne qui découle elle-même de la position verticale de son corps et qui a permis à la fois le développement du cerveau et l'épanouissement des cavités ethmoïdales sinusiennes?

La plupart des anthropologistes modernes n'hésitent pas à affirmer que c'est la *verticalité* qui progressivement a favorisé le développement de notre squelette céphalique.

Il n'est pas douteux en effet qu'en libérant la main la station verticale ait provoqué la régression des *muscles masticateurs* désormais devenus inutiles pour le combat ainsi que pour la préhension de la nourriture et des proies captives.

Il existe à ce sujet une expérience célèbre de mon maître le Professeur Anthony qui en supprimant chez le tout jeune chien des muscles masticateurs importants les muscles temporaux provoqua ultérieurement un élargissement latéral et en hauteur de la boîte crânienne.

Ainsi peut s'expliquer pour les futurs hominiens le rôle de la verticalité dans l'augmentation de volume du crâne.

Or pendant que la main de l'Homme acquerrait la précision et la finesse de l'instrument merveilleux qu'elle devait devenir plus tard la morphologie du crâne et de la face se transformait.

La face grandit en hauteur en largeur et en harmonie ce qu'elle perdait en longueur en épaisseur et en puissance.

C'est ainsi que l'ethmoïde humain libéré de ses contraintes latérales et frontales a pu s'épanouir entre un sinus maxillaire chargé un sinus sphénoïdal et un sinus frontal surélevé.

Dans le même temps le naso-turbinal des Mammifères disparaissait mais il laissait tout de même sa trace par le moyen du monticule de l'aggrégat.

On peut donc se représenter l'évolution des cavités paranasales de l'Homme de la façon suivante. Deux facteurs ont profondément modifié les dispositions ancestrales primitives du complexe maxillo-fronto-sphénoïdal des Primates.

1° L'apparition chez l'Homme d'un appareil pneumatique ethmoïdal très développé et *dynamique dans son expansion*.



FIG. 10. Même section que dans la figure précédente. Toutes les voies de drainage ont été cathétérisées. En arrière, le sinus sphénoïdal débouchant dans un recessus ethmoïdo sphénoïdal par faitement dessiné. En haut, 3 drains placés dans le sinus frontal conduisant, en allant d'avant en arrière : 1° le premier dans une cellule ethmoïdale antérieure, 2° le deuxième ressort par un orifice classique au niveau de la gouttière semi lunaire, 3° le dernier enfin pénètre directement dans le sinus maxillaire.



FIG. 11. La paroi externe des fosses nasales de l'Homme. Cette figure agrandie montre parfaitement la région des gouttières et l'ensemble de la région ethmoïdale antérieure, postérieure et sphénoïdale. Elle démontre d'une façon saisissante le caractère spécifique que de l'ethmoïde humain auquel aucun autre ethmoïde n'est comparable.

2° La disparition progressive chez les grands Primates et définitive chez l'Homme du naso turbinale qui bien que ne faisant pas partie de l'ethmoïde participait d'une façon importante chez certains Mammifères à la pneumatisation de la face en liaison avec le sinus maxillaire et avec le sinus frontal.

En définitive le labyrinthe ethmoïdal par son interposition parfois débordante entre le groupe maxillo frontal et le sinus sphénoïdal a modifié les

rapports primitifs du sinus maxillaire et du sinus frontal, en incorporant progressivement, puis définitivement le sinus frontal à son propre système

On pourrait même avancer que le sinus frontal est une véritable conquête de l'ethmoïde humain

Ainsi s'explique, grâce à l'anatomie comparée et l'embryologie les dispositions spécifiques nouvellement adoptées par les cavités pneumatiques de la face chez l'Homme

L'aspect si particulier des sinus humains revêt une importance telle qu'on peut se permettre de le faire entrer en ligne de compte dans la compréhension évolutive de l'Homme lui-même, dont on pourrait donner la définition suivante

L'Homme est un Primate essentiellement caractérisé par son bipédisme, la verticalité de son corps, le développement de sa boîte crânienne et de son encéphale, et par l'épanouissement de ses cavités paranasales groupées autour d'un labyrinthe ethmoïdal dynamique et parfaitement systématisé

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DISCUSSION

M. Schwarz. Die Formbildung der menschlichen Nebenhöhlen ist von drei Faktoren abhängig: von der Geruchsfunktion, von der ausserordentlichen Entfaltung des

Gehirns bzw. Gehirnschadels und vom aufrechten Gang. Die formale Entwicklung des Siebbeines geschieht beim höheren Säugetier wie beim Menschen nach den gleichen Prinzipien: die Siebbeingänge nach dem Gesetz der asymmetrischen Dichotomie, die der pneumatischen Höhlen durch überstürzte Teilung der Gänge (polymere Bildungen) oder durch adventive Knospung. Das menschliche Siebbein ist demnach kein spezifisch menschliches Organ.

P. Ardouin. Je remercie vivement le Docteur Schwarz d'avoir bien voulu s'intéresser à ma communication.

Toutefois, je dois lui répondre que je ne me suis pas placé, dans mon travail comparatif, sur le plan de la fonction olfactive, mais sur le plan strictement anatomique des cavités pneumatiques creusées dans le squelette cranio-facial.

Or, si tous les Mammifères possèdent une capsule nasale de nature ethmoïdale avec des cornets ethmoïdaux parfaitement déroulés (surtout chez les macrosmatiques), aucun Mammifère ne possède un appareil ethmoïdal aussi étendu et aussi parfaitement systématisé que celui de l'Homme, dont il reste l'apanage exclusif.

A METHOD OF INVESTIGATING THE AIR CONDITIONING MECHANISM OF THE NOSE

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An apparatus has been constructed to deliver air at any temperature and degree of humidity so that the changes in the nasal mucosa may be recorded by means of a tube measuring the pressure in the naso-pharynx. Considerable difficulties have been experienced in the construction and these will be referred to together with some of the results in Man and animals.

In previous communications I have described the air conditioning mechanism of the nose of animals and Man and have expressed the opinion that the main reasons for an efficient system are the requirements of olfaction with secondary concern for the respiratory system. The arguments need not be repeated here.

In an attempt to elucidate the finer details of adaptation to atmospheres of varying temperature and humidity I have during the last few years carried out experiments to determine the changes in the nasal mucosa in response to varying conditions and to this end an apparatus has been constructed to deliver air at any required temperature and humidity to the nose of the subject under examination whether animal or human. The approach is somewhat different to that of many other investigations.

The apparatus consists of a fan driven by a small motor capable of delivering a volume of up to 20 litres a minute at a low pressure. The air is led by wide bore tubes (2.5 cm) to moulded nose pieces for a man or to a mask for animals (Fig. 1).

The current of air passes through inspiratory flap valves of a design similar to those used with tracheostomy tubes and escapes through side tubes fitted with similar expiratory valves. The subject thus inspires and expires air from a constant stream with no resistance to inspiration and practically none to expiration (Fig. 2). Phases of respiration are recorded from a stethograph by means of a tambour and writing arm.

The design is such that there is practically no dead space and marked stagnation of expired air would invalidate results.

The air can be cooled by passing through wide bore copper channels immersed in alcohol containing CO_2 ice. The temperature after passing through two cooling chambers can be brought down to -63°C (-82°F). Since very cold air will not carry moisture the emerging current is almost completely dry and can be delivered to the nose in this state. In its passage from cooling chamber to nose there is a rise in temperature from -60°C to about -40°C .

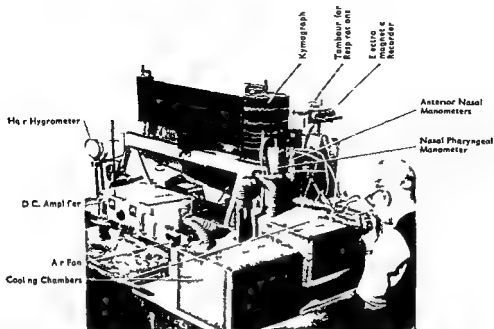


FIG. 1. Apparatus for investigating the response of the nose to changes of temperature and humidity

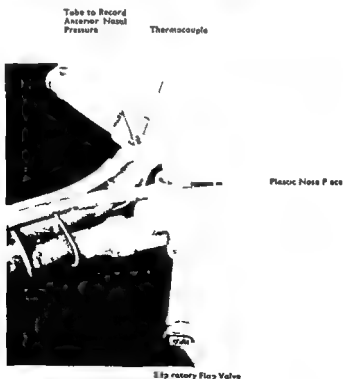


FIG. 2. Nasal connections

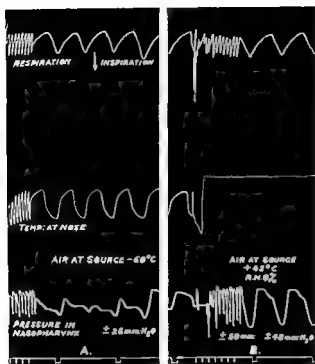


FIG. 3. A subject breathing very cold air with recording of the nasopharyngeal pressure (1) when hot dry air is breathed resistance in the nose increases owing to filling of sinusoids and the nasopharyngeal pressure rises (B). During inspiration the temperature of cold air entering the nose drops while during expiration it rises.

(-40°F) which is sufficiently low to give suitable results and with no apparent danger to the subject under investigation.

Any higher temperature and any degree of humidity is obtained by using room air either at its natural relative humidity or dried by passing through the freezing chambers with subsequent re-warming.

The air current can be heated by passing through a tube with an electric element and can then be either dry or moistened by passing through an electric bottle.

The temperature of air as it reaches the nostril is measured by a fine thermocouple (Fig. 2) connected to a D.C. amplifier and an arm writing on a smoked drum actuated by an armature swinging between electro-magnets (Fig. 1).

The degree of swelling of the nasal mucosa determines the resistance in the nose and this is recorded by a writing point actuated by a water manometer with connection to a mouth piece consisting of an external flap and an up-turned tube held in the mouth above the tongue.

In the case of animals pressure and temperature are measured directly in the trachea by means of a needle of 2 mm bore and a fine thermocouple. The degree of relative humidity can be determined by comparison of two thermocouples one with a wet point.

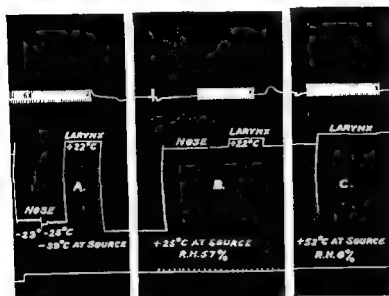


FIG. 4. Air was drawn at 3 strokes per minute with a minute ventilation rate of 8 litres through the nose of a man whose larynx had become separated from the trachea as the result of an accident. Air at a temperature at source of -39°C reaching the nose at -23°C to -26°C is raised after passing through the nose to $+22^{\circ}\text{C}$ at the lower end of the larynx into which a rubber tube was inserted (A).

With air at 25°C R.H. 57%, the temperature below the larynx was $+22^{\circ}\text{C}$ (B) and with hot air at $+52^{\circ}\text{C}$ R.H. 60% the laryngeal temperature was again $+22^{\circ}\text{C}$ (C).

This experiment shows that the nose alone can in the absence of warm expired air raise the temperature of inspired air but not up to body level.

Two upturned fine tubes connected to manometers are fixed to the nose pieces to record anterior nasal pressures but so far they have not been of practical use in recording relative air flow.

A summary of conclusions arrived at concerning the effect of varying atmospheres on the nasal mucosa of man is as follows: (a) swelling of the mucosa was in general less with very cold air than with air at room temperature ($+18^{\circ}$ to $+25^{\circ}\text{C}$); (b) cold air caused less congestion than hot air (Fig. 3); (c) moist air at room temperature caused less swelling than hot dry air; (d) dry room air was associated with greater swelling than with moist air at the same temperature.

The changes in man are not very marked and it would appear that air conditioning for the benefit of the respiratory tract is somewhat fortuitous; the warming and moistening mechanism is primarily concerned with olfaction which in man is feeble.

The noses of animals with keen powers of scent are very much better provided with an apparatus for air conditioning by virtue of branching maxillo-turbinal bodies of great complexity and very extensive area; there is always a direct relation of this efficient humidifying mechanism to the extent of the

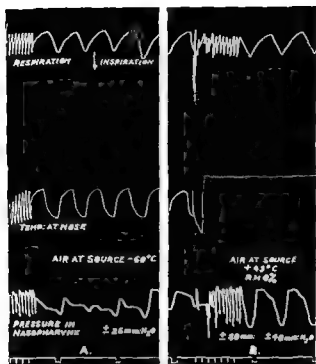


FIG. 3 A subject breathing very cold air with recording of the nasopharyngeal pressure (4) when hot dry air is breathed resistance in the nose increases owing to filling of sinusoids and the nasopharyngeal pressure rises (B) During inspiration the temperature of cold air entering the nose drops while during expiration it rises

(-40°I) which is sufficiently low to give suitable results and with no apparent danger to the subject under investigation

Any higher temperature and any degree of humidity is obtained by using room air, either at its natural relative humidity, or dried by passing through the freezing chambers, with subsequent re-warming

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eliminates any peripheral interference and does not require pre testing of the two ears separately. A standard of integration for normal subjects may be determined as we shall see later. The only difficulty is represented by cases with widely different audiometric thresholds but it may be obviated up to a certain extent by special devices.

The technical description of the apparatus used has been published elsewhere. It will be enough to recall that the message is periodically switched from one ear to the other so that each ear receives one half of the message. The switching rate is variable according to will so that the responses of the subject may be checked at different rates.

The first obvious criticism is that the test avails itself of short sentences, whose integration may depend upon the intellectual level of the testee thereby making the determination of a standard of integration impossible.

Teatini and Demitri in our clinic carried out several experiments in which different testing materials such as trisyllabic words of different grades of difficulty and short and long sentences meaningful and meaningless were presented by the switching technique to three different groups of subjects with widely different scores at the intelligence vocabulary and memory tests. No difference in the integration of the switched message could be revealed in the different groups of subjects for both isolated words and short sentences. Discrimination was always between 90% and 100% at any switching rate. Curiously enough the average score for meaningful sentences was just a little worse than that for meaningless sentences.

Only when using long sentences does the memory and vocabulary of the testee appear to affect considerably the results of the test. Therefore it may be assumed that neither the quality of the message nor the intelligence of the testee have any influence upon the result of the switched speech test when short meaningful sentences are used. From this point of view the test may be considered as reliable.

The second criticism of the method is more substantial. According to Colin Cherry & Taylor when the switching rate approaches four per second a definite dip of discrimination appears which is attributed to mental delay. According to these authors the necessary time for the attention to shift from one ear to the other cannot keep pace with the switching rate so that the attention lags behind and a part of the message is lost. By further increasing the switching rate the discrimination improves again but this is not due to compensation of the delay the testee suppresses unconsciously the half messages coming from one ear limiting himself to integrating the interrupted message in the other ear. The mathematical integration of the right flank of the dip and the discrimination curve for interrupted speech at increasing interruption rates seems to lend support to the hypothesis. If this is correct then the switched speech test as a test of binaural summation must be denied any value.

As a matter of fact the hypothesis of Colin Cherry & Taylor could not be proved.

(1) No dip could be demonstrated for ordinary output speed at any switching rate

(2) A dip between three and five switches per second could be demonstrated using accelerated speech, but there was never an analogy between the right flank of the dip and the discrimination curve of the same subject for the interrupted speech

(3) Many pathological cases show no dip whatsoever at the switched speech test, while their discrimination of the interrupted speech is bad at any interruption rate

As a consequence, the conclusion may be drawn that a real summation of the two halves of the message takes place at any switching rate and that the dip, when present only means that probably some unknown synaptic rhythm is temporarily put out of gear when the switching rate is between three and five per second

The increasing rate of switching does not prevent summation from coming into action and deficiencies in discrimination must be ascribed to troubles of binaural integration

We feel that the switched speech test may prove an effective tool in the diagnosis of brain stem pathology, vs cortical or peripheral lesions

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DISCUSSION

E. Bocca (Reponse) Je suis tout à fait d'accord avec M. Hennebert que l'apparition de « dips » plus ou moins importants dans le test de la voix commutée est liée à la difficulté du message, mais ce que nous avons cherché d'obtenir a été justement d'élaborer un matériel verbal qui n'influence pas les résultats du test par rapport à l'intelligence du sujet. Ce que nous intéresse n'est pas d'étudier les variations des résultats selon le matériel et l'intelligence du sujet, mais, en nous basant sur ces études, de mettre à point un test « standard » valable dans les conditions les plus différentes afin de nous en servir comme moyen d'investigation clinique

MODIFICATIONS DE L'ADAPTATION AUDITIVE D'ORIGINE CENTRALE PAR INTERFÉRENCES SENSORIELLES

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L'adaptation auditive monaurale étudiée avec la méthode du temps de disparition de la perception du seuil présente des modifications remarquables chez le sujet normal par l'effet soit de stimuli sonores présentés en même temps à l'oreille controlatérale soit d'afférences visuelles simultanées.

Les caractères des modifications de l'adaptation par ces interférences sensorielles sont nettement altérés chez des patients atteints par différentes lésions du système nerveux central.

Les recherches électrophysiologiques ainsi que psychoacoustiques semblent confirmer l'hypothèse que le comportement de l'adaptation soit lié d'une façon prépondérante à l'activité de structures nerveuses de l'appareil auditif. Les premières indiquent unanimement l'absence de décroissements des potentiels microphoniques cochléaires par l'effet de tons continus de faible intensité (Maniagrazzini, Pellegrini, Pestalozza, Shumizu, Konsho, Nakamura, Gisselsson & Sorensen).

Une diminution des réponses électriques par suite de l'exposition à la lumière blanche ou à des tons continus a été observée par divers auteurs tout le long du cours des voies acoustiques à partir du nerf cochléaire jusqu'à l'écorce temporale.

Quoique se manifestant par diverses modalités selon les niveaux étudiés, cette décroissance de l'activité électrique de la voie acoustique a été constatée pour les fibres du nerf VIII (Galambos & Davis), pour les noyaux cochléaires (Galambos, Sheatz & Vernier), pour les tubercules quadrijumeaux inférieurs et les corps geniculés médiaux (Galambos, Gross & Thurlow), pour la zone acoustique primaire du lobe temporal (Brumer, Whitfield).

Au niveau des noyaux cochléaires on a encore observé que les réponses électriques à des tons continus peuvent être modifiées par la présentation simultanée de stimulus visifs, olfactifs ou douloureux ou bien par excitation électrique de la substance réticulaire (Hernandez Peon, Scherter, Jouve).

Quant à ce qui concerne les expériences psychoacoustiques, on doit noter que le déterminisme de l'adaptation auditive est de nature différente selon la méthode employée. En effet l'évaluation monaurale du reste d'adaptation successive à un ton bref proposée par Lüscher & Zwislowski est probablement de nature périphérique étant donné qu'elle n'est pas modifiable par des

stimuli contralatéraux et s'accroît avec l'augmentation de l'intensité de l'impulsion stimulante selon une courbe en forme exponentielle.

Le déterminisme de l'adaptation étudiée avec cette méthode serait lié en effet selon Langenbeck & Kietz à des facteurs mécaniques de déphasage de vibration entre la membrane basilaire et la membrane de Reissner tandis que selon Luscher & Zwislowski, G. de Mare & V. Dishoeck il serait dû à des phénomènes de polarisation de membrane au niveau des récepteurs ou des achevements nerveux.

Mais si l'on considère les méthodes qui étudient la perte de perception pour un ton continu il semble que l'adaptation auditive induite de cette façon soit liée à l'activité des structures de la voie acoustique centrale.

Cette considération apparaît valable pour les trois méthodes employées pour évaluer ce type d'adaptation (balancement binaural périodique par stimulateur, seuil sous masque, temps d'épuisement du seuil).

En effet l'adaptation étudiée avec le balancement binaural par stimulateur proposée par Hood n'est plus apparente si l'on stimule l'oreille non soumise à une adaptation avec rumeur thermique (Montegazzini, Pellegrini & Pestalozza) et elle est donc en rapport avec des phénomènes inhibitoires centraux.

En outre l'adaptation à un ton pur en présence d'un son de masque apparaît indépendante de l'intensité du ton à l'examen (Thwing, Feldmann) tandis qu'on ne peut pas percevoir une perte de perception pour le ton pur due à une exposition prolongée à un bruit de masque d'intensité inférieure à 80-90 db (Egan, Lugh, Pennetta & Pinto). De tels faits sont dus, selon Pirodda, à un différent « pattern » d'excitation des deux stimulus différents au niveau des voies acoustiques centrales. Le temps d'épuisement du seuil enfin a paru alterable par des substances modificatrices du métabolisme des cellules nerveuses (comme l'isopropylamine (Roberto & Pollice)) ou bien par des produits pharmaceutiques qui modifient l'activité du système nerveux central comme le bromure et la caféine (Temkin & Sheikhan). Des modifications soit du type inhibitoire soit comme facilitant le temps d'épuisement du seuil pour les tons purs ont été constatées au moyen des stimulus acoustiques contralatéraux (bruit blanc continu ou interrompu, listes de chiffres que le sujet devait transcrire) dans des recherches effectuées dans notre clinique (Brunetti, Hahn & De Michelis). Le caractère diphasé de ces modifications était en rapport du fait que tandis que la plupart des sujets avec seuil auditif normal présentait un temps d'épuisement du seuil d'au moins 60 sec un pourcentage inférieur (20%) de sujets apparemment normaux présentait un temps d'épuisement du seuil nettement inférieur à la minute.

Tandis que dans les premiers sujets la stimulation acoustique contralatérale provoquait une diminution du temps d'épuisement, dans les seconds la même expérience déterminait une prolongation de la perception du ton adaptant. Des stimulations intenses visuelles (S.I.I.) employées dans la routine EEG déterminaient au contraire dans les deux groupes une très nette réduction du temps d'épuisement du seuil.

Des observations ultérieures effectuées dans notre clinique par Hahn et

collaborateurs sur des sujets au seuil auditif normal et à temps bref d'épuisement du seuil ont démontré que la perte de perception à cause d'un bruit blanc présente au seuil quoique étant moindre que celle due à de simples tons (comme l'avait déjà noté Carterette) n'est pas proportionnelle à celle pour les tons purs indiquant par conséquent que l'adaptation étudiée de cette façon n'était pas uniquement en rapport avec l'activité des récepteurs et avec la vibration de la membrane basilaire (Hahn).

Enfin la durée de la perception du seuil du bruit blanc semblait susceptible de modifications par des tons purs contralatéraux présentes avec une intensité de 30 db avec de plus grandes variations si les tons interférents étaient de fréquence grave (Hahn) phénomène analogue à ce qui a été décrit par Galambos et par Gross & Thurlow au cours de recherches électrophysiologiques.

La méthode du temps d'épuisement du seuil soit à cause de sa simplicité d'emploi soit pour sa sensibilité à l'égard de différents types d'interférences centrales est résultée comme étant particulièrement indiquée comme élément sémiologique des lésions de la voie acoustique centrale.

Malgré ces caractéristiques la méthode du temps d'épuisement du seuil a été utilisée par peu d'auteurs seulement pour l'étude des altérations de la voie acoustique centrale et généralement sur un nombre limité de cas.

La plupart des observations concerne des patients affectés de tumeurs de l'angle ponto cérébelleux (Rieger & nos Jerger Carhart & Lassmann Yantis Pestalozza) étudiées différemment soit avec la méthode de Carhart (présentation d'un ton continu au niveau de seuil recherche de l'intensité nécessaire pour que le ton soit perçu pendant 10 sec) soit avec l'audiomètre de Bekesy (ton continu au seuil présenté avec modulation d'intensité et enregistrement graphique de la sensation de la part du sujet) soit enfin avec la méthode proposée par Pestalozza (présentation d'un ton continu pendant 3 min à l'abord du seuil et successivement augmenté de 3 db chaque fois que le sujet perd la perception).

Chez tous les patients il a été observé que le temps d'épuisement du seuil se présente sous une forme très réduite (inférieure à 30 sec) en outre une intensité très élevée est nécessaire pour que la perception du ton continu persiste longtemps (de 30 à 60 db sur le seuil). Dans quelques cas à l'intensité maximum consentie par l'audiomètre la perception du ton disparaissait après quelques secondes (Pestalozza Jerger Carhart & Lassmann).

Quant à ce qui concerne d'autres lésions du système nerveux central les observations sont encore plus rares. On doit rappeler le cas de Jatho concernant une tumeur du tronc cérébral dans lequel on avait noté de graves altérations du temps d'épuisement de seuil ainsi que la casuistique de Pestalozza. Cet auteur a relevé de faibles modifications de l'adaptation dans les lésions du tronc cérébral (2 cas) résultats en contraste dans des cas d'épilepsie temporaire avec altération du temps d'épuisement du seuil parfois omolaterales parfois contralatérales à la lésion et de légères modifications de la norme dans des cas de néoplasmes temporaires.

La méthode pour la recherche du temps d'épuisement du seuil employée

stimuli contralatéraux et s'accroît avec l'augmentation de l'intensité de l'impulsion stimulante selon une courbe en forme exponentielle.

Le déterminisme de l'adaptation étudiée avec cette méthode serait lié en effet selon Langenbeck & Kietz à des facteurs mécaniques de déphasage de vibration entre la membrane basilaire et la membrane de Reissner tandis que selon Luscher & Zwisłowski G de Mare & V Dishoeck il serait dû à des phénomènes de polarisation de membrane au niveau des récepteurs ou des achèvements nerveux.

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La méthode du temps d'épuisement du seuil soit à cause de sa simplicité d'emploi soit pour sa sensibilité à l'égard de différents types d'interférences centrales est résultée comme étant particulièrement indiquée comme élément semiologique des lésions de la voie acoustique centrale.

Malgré ces caractéristiques la méthode du temps d'épuisement du seuil a été utilisée par peu d'auteurs seulement pour l'étude des altérations de la voie acoustique centrale et généralement sur un nombre limité de cas.

La plupart des observations concerne des patients affectés de tumeurs de l'angle ponto-cérébelleux (Reger & Kos, Jerger, Carhart & Lassmann, Yantis, Pestalozza) étudiées différemment soit avec la méthode de Carhart (présentation d'un ton continu au niveau de seuil, recherche de l'intensité nécessaire pour que le ton soit perçu pendant 60 sec) soit avec l'audiomètre de Bekesy (ton continu au seuil présenté avec modulation d'intensité et enregistrement graphique de la sensation de la part du sujet) soit enfin avec la méthode proposée par Pestalozza (présentation d'un ton continu pendant 3 min d'abord au seuil et successivement augmentée de 5 db chaque fois que le sujet perd la perception).

Chez tous les patients il n'a été observé que le temps d'épuisement du seuil se présente sous une forme très réduite (inférieure à 30 sec) en outre une intensité très élevée est nécessaire pour que la perception du ton continu persiste longtemps (de 30 à 60 db sur le seuil). Dans quelques cas à l'intensité maximum consentie par l'audiomètre la perception du ton disparaissait après quelques secondes (Pestalozza, Jerger, Carhart & Lassmann).

Quant à ce qui concerne d'autres lésions du système nerveux central les observations sont encore plus rares. On doit rappeler le cas de Jatho concernant une tumeur du tronc cérébral dans lequel on avait noté de graves altérations du temps d'épuisement de seuil ainsi que la casuistique de Pestalozza. Cet auteur a relevé de faibles modifications de l'adaptation dans les lésions du tronc cérébral (3 cas) résultats en contraste dans des cas d'épilepsie temporale avec altération du temps d'épuisement du seuil parfois unilatérales parfois contralatérales à la lésion et de légères modifications de la norme dans des cas de néoplasmes temporaux.

La méthode pour la recherche du temps d'épuisement du seuil employée

stimuli contralatéraux et s'accroît avec l'augmentation de l'intensité de l'impulsion stimulante selon une courbe en forme exponentielle.

Le déterminisme de l'adaptation étudiée avec cette méthode serait lié en effet selon Tjengbeek & Kietz à des facteurs mécaniques de déphasage de vibration entre la membrane basilaire et la membrane de Reissner tandis que selon Luscher & Zwislowski & de Mare & V. Dishoeck il serait dû à des phénomènes de polarisation de membrane au niveau des récepteurs ou des achevements nerveux.

Mais si l'on considère les méthodes qui étudient la perte de perception pour un ton continu il semble que l'adaptation auditive induite de cette façon soit liée à l'activité des structures de la voie acoustique centrale.

Cette considération apparaît valable pour les trois méthodes employées pour évaluer ce type d'adaptation (balancement binaural périodique per stimuloire, seuil sous masque, temps d'épuisement du seuil).

En effet l'adaptation étudiée avec le balancement binaural per stimuloire proposée par Hood n'est plus apparente si l'on stimule l'oreille non soumise à une adaptation avec tumeur thermique (Montegazzini, Pellegrini & Pestalozza) et elle est donc en rapport avec des phénomènes inhibitoires centraux.

En outre l'adaptation à un ton pur en présence d'un son de masque apparaît indépendante de l'intensité du ton à l'examen (Thwing, Feldmann) tandis qu'on ne peut pas percevoir une perte de perception pour le ton pur due à une exposition prolongée à un bruit de masque d'intensité inférieure à 80-90 db (Egan, Jugh, Pennetta & Pini). De tels faits sont dus selon Pirodda à un différent « pattern » d'excitation des deux stimulus différents au niveau des voies acoustiques centrales. Le temps d'épuisement du seuil enfin a paru altérable par des substances modificatrices du métabolisme des cellules nerveuses (comme l'isopropylamine (Roberto & Pollice)) ou bien par des produits pharmaceutiques qui modifient l'activité du système nerveux central comme le bromure et la caféine (Femkin & Sheikhan). Des modifications soit du type inhibitoire soit comme facilitant le temps d'épuisement du seuil pour les tons purs ont été constatées au moyen des stimulus acoustiques contralatéraux (bruit blanc continu ou interrompu, listes de chiffres que le sujet devait transcrire) dans des recherches effectuées dans notre clinique (Brunetti, Hahn & De Michelis). Le caractère diphasé de ces modifications était en rapport du fait que tandis que la plupart des sujets avec seuil auditif normal présentait un temps d'épuisement du seuil d'au moins 60 sec, un pourcentage inférieur (20%) de sujets apparemment normaux présentait un temps d'épuisement du seuil nettement inférieur à la minute.

Tandis que dans les premiers sujets la stimulation acoustique contralatérale provoquait une diminution du temps d'épuisement, dans les seconds la même expérience déterminait une prolongation de la perception du ton adaptant. Des stimulations intenses visuelles (S.I.I. employées dans la routine I.L.G.) déterminaient au contraire dans les deux groupes une très nette réduction du temps d'épuisement du seuil.

Des observations ultérieures effectuées dans notre clinique par Hahn et

faibles de l'adaptation auditive pour cela les modifications d'activité dans les voies efferentes cochleaires doivent avoir une importance notable dans ces cas là

Dans les cas de lésions du tronc cérébral l'adaptation auditive est altérée seulement lorsque des phénomènes dégénératifs sont en acte phénomènes intéressant directement les premiers relais des voies acoustiques ou bien des lésions de tumeurs qui détruisent les noyaux cochleaires provoquant aussi une hypoacousie réceptive

Quand la lésion tumorale n'intéresse pas directement la zone des noyaux cochleaires le temps d'épuisement du seuil ne présente pas de modifications et d'une façon analogue à ce qui a été observé dans les scléroses à plaques il n'est pas susceptible d'être modifié par des stimulus acoustiques contralatéraux ou bien par des stimuli visuels

Ce comportement caractéristique de certaines tumeurs du tronc cérébral et des scléroses à plaques pourrait être mis en rapport dans le cas de sclérose multiple avec des phénomènes d'excitation provoqués par le procédé de démyélinisation analogues à ceux qui engagent l'hyperreflexie vestibulaire. Cependant une telle hypothèse peut tomber devant des observations faites sur le comportement analogue du phénomène comme il est démontré dans le cas de tumeurs. On peut donc supposer que chez ces patients la lésion pourrait déterminer un obstacle aux impulsions interférentes qui d'une oreille arrivent à la structure des voies acoustiques effectives pour la stimulation adaptante contralatérale

La symptomatologie auditive chez les malades atteints de lésions extrapyramidales apparaît caractérisée par un seuil normal mais donne une adaptation pathologique avec des crises graves aggravées par des stimulations acoustiques contralatérales spécialement si elles sont envoyées en interruption rythmique. Ces symptômes semblent être caractéristiques aussi des lésions vasculaires sous-corticales profondes et de quelques épileptiques de type sous-cortical sans altérations EEG selon ce qui a été observé dans notre expérience ils semblent donc prêter à une participation des corps géniculés moyens et des structures thalamiques

Actuellement il n'est pas encore possible de donner une explication pathogénétique plausible même hypothétique sur la plus grande action interférente des stimulus acoustiques interrompus

Aussi les épileptiques focaux temporaux présentent des altérations de l'adaptation et se différencient des sujets à forme comitiales sans foyer relevables EEG avec adaptation auditive normale. Contrairement à toute supposition l'altération de l'adaptation chez les épileptiques focaux n'est pas croisée par rapport au côté de la lésion. Ceci peut être probablement en rapport soit avec une existence possible de foyers épileptogènes contralatéraux latents soit avec la différence action inhibitrice ou facilitante des impulsions provenant des mêmes zones épileptogènes

Les altérations contralatérales de l'adaptation relevées dans les lésions vasculaires hémisphériques et dans les cas de tumeurs temporales résultent

être en accord avec le croisement des voies acoustiques. Dans les lésions temporales, les interférences déterminées avec la méthode des listes de chiffres provoquent des modifications plus grandes de la rumeur blanche en rapport avec un effort d'attention plus considérable requis pour la compréhension du test verbal.

L'altération constante de l'adaptation dans les sujets presbycusiques dans lesquels le recrutement est toujours absent donne une confirmation ultérieure à l'importance du composant central dans les troubles de la fonction auditive de l'âge senile.

Pour ce qui a trait à la validité des différents tests d'interférence adoptés, les épreuves basées sur des stimuli acoustiques contrôlés paraissent plus significatives que lorsqu'elles se produisent avec la S. L. I. Cette dernière en effet constitue probablement un stimulus trop intense et semble altérer trop gravement le mécanisme de l'adaptation auditive dans tous les types de lésions étudiés.

Les différents types d'interférence (rumeur blanche continue, rumeur blanche interrompue, liste de chiffres) ont respectivement une plus grande importance semiologique élective au fur et à mesure que l'on monte des zones plus caudales de la voie acoustique aux structures plus rostrales. Les effets de ces interférences sur l'adaptation acoustique centrale se maintiennent constants et caractéristiques même en présence d'altérations de l'adaptation dues à l'existence de surdité simultanée périphérique.

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EXPERIMENTS ON BINAURAL HEARING

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From the Audiology Institute (Head Prof H. C. Huizing) of the University Ear, Nose and Throat Clinic (Head Prof Eelco Huizinga), Groningen

The authors report upon comparative measurements of articulation score in monaural and binaural stimulation of normal ears when stimulated by two narrow bands of coherent filtered speech.

The results indicate that under certain conditions it is to be expected that perceptively impaired ears with sloping threshold audiograms will manifest a substantially better intelligibility for binaural selective amplification than in the case of an equivalent monaural stimulation.

In the Vienna meeting of the Collegium ORLAS last year we reported on the discriminative function of impaired ears within a limited band of frequencies. We measured the articulation score of filtered speech containing only components in the lower, the medium or the treble part of the pitch range. In this way insight may be obtained as to a patient's partial or band discrimination ability.

The results showed that in certain cases of hearing loss, e.g. with a 60 db high tone loss, the discriminative function in this high tone region was fully preserved, provided that sufficient amplification was used. In other cases of high tone deafness, however, the discriminative ability was found to be underdeveloped or even undeveloped by lack of adequate stimuli in early childhood or it had been gradually lost in the course of a progressive type of impairment.

Consequently this variability of results shows that there cannot exist a fixed relationship between a certain value of threshold loss and the loss of speech reception ability involved. Therefore not only routine speech audiometry but also a more analytic testing of band discrimination can give useful supplementary information about a patient's hearing function.

The increasing tendency to prescribe binaural hearing aids emphasizes the future importance of full knowledge as to the patient's discriminative functions in each ear separately as well as combined. In fact the ultimate result of binaural fitting may also depend on the development of an interaural discriminative cooperation. Selective amplification adapted to the distribution of residual discrimination over the pitch range may be helpful in attaining a successful fitting.

In this paper we want to deal with some experiments on normal ears in relation to the well known sloping type of threshold audiogram (Fig. 1). When the slope of such an audiogram exceeds say 10 db/octave we may expect

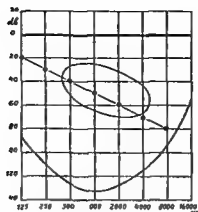


FIG. 1. Threshold audiogram with pre-laminant high tone deafness slope 10 db/octave

the beginning of masking of the essential overtones by the relatively strong low tones. This reduces the quality of the received speech signals so that its intelligibility is substantially lowered.

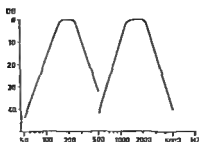
By the following experiment the sloping audiogram of the impaired ear was imitated for the normal ear. Normal speech was processed by means of two one octave filters with frequency bands of 140–280 Hz and 1128–2256 Hz.

The corresponding filter characteristics are given in Fig. 2. In order to keep the processed speech samples coherent a stereophonic tape recorder was used for recording the two filter outputs. By means of a switching arrangement these two samples of coherent speech could be presented each to a single ear or both to the same ear; the intensity levels could be changed at will (Fig. 3). In order to imitate the sloping audiogram the intensity of the low tone band was taken substantially higher than the intensity of the high tone band.

Using P.B. lists the words in the low frequency band were all unintelligible for the normal ear, whereas for the high band an articulation curve could be made as given by Fig. 4. This curve shows that the 50% articulation level is passed at 34 db intensity.

Now a comparison was made as to the articulation scores for P.B. lists for two different ways of proffering these two coherent speech patterns: monaurally vs. binaurally. The result follows from Fig. 5 for the case of a fixed low band intensity of 75 db. We see from this figure that in the case of monaural presentation of both stimuli a 26 db intensity level for the high tone band is needed to reach the 50% articulation level, whereas in binaural stimulation 12 db suffices. Or, to say it the other way, in the monaural case the high tone band should be 14 db more intensive to reach the same intelligibility.

When we relate this result to the slope of a threshold audiogram we may say that in the binaural case of presentation this slope may be $14/3$ (three octaves difference of the two bands) or almost 5 db/octave steeper as compared to the monaural case before masking of the high tone region becomes essential.



110 2 Frequency characteristics of the two band pass filters used

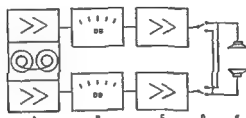
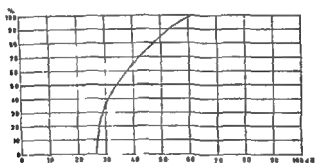
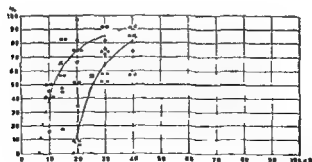


Fig 3 Stereophonic tape recorder and electric circuit for monaural or binaural presentation



110 4 Articulation curve of the higher octave sample for normal ears



110 5 Binaural (curve A) and monaural (curve B) articulation scores for a fixed 75-dB intensity level of the lower band and various intensity values of the higher band (re international standard level)

From this experiment we may conclude that in binaural fitting separate presentation of low and high tones permits a 5 db/octave steeper threshold audiogram before the same amount of discrimination loss is to be expected provided that the patient's discriminative function in the high tone region as well as his loudness function have remained normal

Oostersingel 18^a Groningen

DISCUSSION

E. Bocca Mr Huizing has quite rightly focussed attention upon the importance of binaural summation phenomena in the correction of hearing losses by means of binaural hearing aids. What seems to be important is that even in cases of apparently symmetric sloping audiograms one cannot assume that the same type of amplification in both ears will necessarily bring the best summation results.

Binaural adaptation of hearing aids may become much a wider field for investigation than it has been suspected to be before.

DE QUELQUES PARTICULARITES PHONETIQUES DE L'AUDITION DE LA PAROLE

P. MOUMIER Kuhn et J. C. LAFON¹
Lyon France

Les aspects phonétiques de la perception de la parole sont étudiés à des intensités variables et en présence de bruit, chez l'enfant, l'adulte et le sourd. Les résultats sont confrontés avec la valeur d'information et la fréquence statistique des phonèmes utilisés en français. Ces comparaisons permettent d'entrevoir certaines lois qui régissent le choix inconscient des phonèmes d'une langue.

La conception de la parole que peut avoir un clinicien repose essentiellement sur les éléments de son émission et de sa réception : les défauts d'articulation objectivent la perturbation d'un système moteur ; les surdités, celles d'un système sensoriel. Les variations de la composition du message transmis deviennent ainsi l'expression d'une altération d'un système physiologique. Si le clinicien s'intéresse au langage, c'est pour déceler à travers les déformations individuelles l'indice d'une lésion déterminée dans le cadre d'une semiologie clinique.

On a tendance à oublier que le signifiant représenté par les images acoustiques ne peut exister qu'en fonction d'un système abstrait dont les lois et la structure sont très éloignées de la clinique. On conçoit cependant d'un point de vue théorique que l'audition et la phonation soient intimement liées au langage, que ce dernier soit tributaire des qualités physiologiques des organes transmetteurs et récepteurs, mais il est difficile de dégager les éléments d'une confrontation, car si l'expression acoustique est bien un domaine commun au linguiste et au clinicien, les modalités d'étude et de description, leurs préoccupations sont suffisamment éloignées pour qu'il ne puisse y avoir une terminologie et une systématique communes.

A l'heure où la phonétique devient de plus en plus expérimentale et où la linguistique s'efforce de répondre à sa vocation de science du langage, le physiologiste doit participer dans l'étroite activité linguistique qui lui est dévolue à cette construction en apportant les matériaux glanés dans une pratique quotidienne.

En utilisant des éléments de phonétique acoustique et de phonétique statistique, nous voudrions essayer de montrer que cette confrontation entre langue et pathologie permet d'entrevoir la nécessité et le sens d'un dialogue plus approfondi. L'otologie confine à la phonologie.

Deux ordres de matériaux retiendront notre attention : d'un côté, des élé-

¹ Attaché de Recherche au C.N.R.S.

From this experiment we may conclude that in binaural fitting separate presentation of low and high tones permits a 5 db/octave steeper threshold audiogram before the same amount of discrimination loss is to be expected provided that the patient's discriminative function in the high tone region as well as his loudness function have remained normal

Oostersingel 15^e Groningen

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P. MOUVIER ELKH et J. C. LAFONT
Lyon France

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Deux ordres de matériaux retiendront notre attention : d'un côté

ments d'intelligibilité phonétique fournis par les réponses de sujets adultes normaux ou sourds et d'enfants atteints de retard du langage d'un autre côté les données de cinq statistiques phonétiques permettront de rapprocher les études expérimentales des spéculations abstraites

1 L'intelligibilité phonétique

Si l'intelligibilité phonétique peut se mesurer à voix nue la distance faisant office d'assourdissement comme le pratiquaient l'Abbé Rousselot en phonétique et encore récemment l'otologiste en clinique son étalonnage précis est malaisé. Aussi le secours des appareils électroniques est-il nécessaire à la mesure de la compréhension de la parole l'intensité pouvant alors être parfaitement contrôlée. Par le biais des amplificateurs on peut d'autre part fournir des « bruits » pour compliquer le message et rendre son identification plus malaisée. Le terme « bruit » étant dans cette phrase utilisé suivant la définition des télécommunications il correspond à une gêne que ce soit un bruit vrai un filtrage des interruptions rythmiques etc.

Pour mesurer cette compréhension nous devons choisir des messages présentant une structure linguistique précise. Nous avons utilisé trois séries de mots. Pour l'individu normal entraîné nous avons choisi des logatomes de deux phonèmes.

Pour les autres études nous nous sommes servi des listes du test phonétique chez l'enfant la liste de quarante mots de trois phonèmes intitulée

« Balayage phonétique » (Cahier C.F.A. n° 6 Paris 1967) en apportant dans le dépouillement les corrections statistiques nécessaires. chez l'adulte des listes permettant de mieux différencier les difficultés de compréhension en les opposant aux surdités listes récemment définies (CR Ac Sc novembre 1969) et dont voici les bases.

Le test phonétique

Lorsque nous entendons un mot nous l'identifions à l'aide de ses constituants élémentaires les phonèmes suivant le souvenir que nous avons d'un mot acoustiquement semblable. Certains phonèmes sont indispensables à la reconnaissance ce sont des *phonèmes caractéristiques*. Si on ne les perçoit pas il est pratiquement impossible de reconstituer le mot on a le choix entre une série de mots qui ont à ce phonème près la même structure acoustique. Cette série constitue une famille ce sont ces familles que nous avons utilisées. Nous avons choisi les séries contenant comme phonèmes variables des phonèmes confondus entre eux à l'audition. C'est ainsi que le support « ar » a été choisi. m de mar peut être confondu avec n et v v peut être confondu avec f j ch etc et ces phonèmes donnent la liste de mots « mar nar lar rar var far » jar » char lar dar gar etc. Une confusion auditive engendrera un mot faux ma (trouvant à une autre endroit de la liste comme mot test. Tous les

phonèmes courants de la langue française sont explorés par deux listes de cinquante mots construits suivant ce principe. Évitant l'écueil de la disponibilité des mots de la mémorisation des mots de la liste et de leur connaissance ces listes peuvent être utilisées plusieurs fois de suite. Le sujet n'a pas la possibilité de supplier un défaut de l'audition à condition que l'ordre de présentation soit imprévisible. On peut ainsi connaître comment l'individu perçoit chaque phonème et quelle forme il lui donne.

Les modalités de transmission de la parole sont un des facteurs de la compréhension. La parole trop faible ou trop forte, la parole masquée par du bruit sont plus difficiles à comprendre que la parole émise normalement dans un lieu silencieux. En rendant le message difficile à comprendre et en chiffrant cette difficulté on peut ainsi tester la capacité d'intégration auditive.

Pour des raisons pratiques nous utilisons la transmission à travers un audiomètre de la même liste suivant quatre modalités de difficulté croissante. Première émission à 70 db (I) (l'optimum de compréhension se situe à 10 db) qui nous sert de référence. On note le nombre de phonèmes testés mal reproduits dans la liste et quelles sont les confusions. Deuxième émission à 90 db (II) le nombre de phonèmes mal reproduits augmente s'il en existait à 70 db. Troisième émission à 90 db avec un bruit (dent de scie) de 75 db (III) quatrième émission à 90 db avec 90 db de bruit (IV). L'identification demande une bonne attention même pour un sujet normal.

Si l'on soustrait le chiffre obtenu à 70 db des autres et que l'on reporte les résultats sur un graphique on met en évidence la qualité de reconnaissance auditive du sujet son niveau d'intégration phonétique. Cette soustraction permet de rendre intelligible la connaissance de la langue les difficultés motrices d'élaboration du mot ou d'articulation qui sont les mêmes quel que soit le mode de réception sensorielle.

Avant d'éliminer l'influence des facteurs moteurs de la mémoire et de la composition linguistique il reste encore les déformations provoquées par l'oreille. Celles-ci ont le même pouvoir de distorsion à une intensité constante d'émission quel que soit le bruit surajouté. Entre II, III et IV il n'y a que le facteur compréhension pour amener une aggravation significative du nombre de phonèmes mal répétés en particulier entre II et IV lorsque il y a une forte surdité.

Ces listes présentent l'avantage d'une excellente précision diagnostique entre surdité et intégration. Elles ont le défaut de n'offrir qu'un nombre de transitions phonétiques limité (voir troisième série d'études) donc d'aggraver statistiquement l'identification de quelques phonèmes.

II. Études expérimentales

1) Les premiers résultats concernent le dépouillement de dossiers de la consultation d'enfants atteints de troubles de l'audition et du langage à la Clinique O.R.L. Universitaire de Lyon. Nous n'avons retenu que les enfants dont l'intelligibilité a été suffisamment explorée pour pouvoir être chiffrée.

Le « r » et le « m » ont des caractéristiques inversées par rapport à l'enfant
 « r » 24%-4% « m » 21%-51%

On peut penser, surtout pour le « r » à des possibilités de reconnaissance par le sourd adulte meilleure que celle du sourd enfant qui ne connaît le phonème qu'à travers sa surdité

Inversement comme chez l'enfant le « on » est moins bien perçu par le sourd (57%-27%) de même que le « m » (72%-21%) dont la différence est plus nettement marquée. Notons encore le phonème « æ » (non testé chez l'enfant) (27%-05%) et le « an » (7,2%-38%) dont les chiffres sont inversés entre surdité et intégration par rapport à l'enfant. Les autres variations ne se distinguent pas des marges d'erreurs.

Le rôle essentiel de la composition acoustique des phonèmes apparaît plus nettement chez l'adulte que chez l'enfant : les qualités acoustiques des phonèmes expliquent les difficultés de compréhension.

3) La troisième série de résultats concerne des individus entraînés à une audition liminaire. On a cherché à établir quelle pouvait être l'interaction des juxtapositions phonétiques dans l'intelligibilité d'un complexe de type syllabique à intensité d'émission constante.

Cette étude difficilement réalisable en clinique, du fait de l'importance et de la durée des épreuves à faire, nous donne des résultats intéressants : l'intelligibilité des phonèmes en fonction de leur structure acoustique (sujets entraînés) et des juxtapositions phonétiques.

Les phonèmes les plus difficiles à percevoir appartiennent pour les consonnes en majorité aux sifflantes. Par ordre : « f », « v », « z », « k », « s », « d ». Pour les voyelles tout dépend de l'aperture : les voyelles fermées sont plus touchées. Par ordre : « u », « ou », « on », « i », « un », bien que le « e » fermé soit le plus facile à reconnaître : peut-être est-il plus intense ?

L'influence des consonnes sur l'intelligibilité des voyelles est faible : « f » et « gn » rendent plus difficile l'identification ; contrairement à « l », « s », « z » et « ch ».

Les voyelles influencent beaucoup plus les consonnes. Lorsque la voyelle suit la consonne on constate que la fermeture fait apparaître une difficulté d'identification. Par ordre de difficulté : « i », « e », « on », « ou », « u » sans exception puisque le « e » ne se comporte pas différemment des autres phonèmes. Les voyelles ouvertes rendent plus facile l'intelligibilité.

ADDITION AU SYLLABE

Par ordre de difficulté

Consonnes f v z k s

Voyelles u on ou i un

Phonèmes majoritaires de la difficulté

Consonnes l gn n m r

Voyelles i e on ou u

III Etude phonétique statistique

Les difficultés de compréhension paraissent se expliquer assez facilement par le spectre du phonème par les intensités relatives dans la suite du dis

TABLEAU 2

Phonèmes	Français écrit	Français parlé	Français grammatical	Phonèmes caractéristiques
a patte	5 30	8 1	8 37	4 0
m pâte	0 63	0 2	—	0 5
m rate	3 11	5 3	5 9	3 6
m lés	7 55	6 5	5 0	1 8
i vie	0 59	—	—	—
i si	5 77	5 6	5 7	3 0
i bien	—	1 0	—	—
m instable	4 90	4 9	9 4	—
m rate	5 16	—	—	—
œ œuf	0 45	0 3	0 9	0 6
œ mieux	0 55	0 6	1 0	0 8
û but	2 71	2 0	1 8	1 1
û huis	—	0 7	—	—
m mauve	0 93	1 7	1 0	1 5
o port	2 21	1 5	1 6	1 9
u mou	2 08	2 7	—	—
w oui	0 69	0 9	1 8	2 2
w loin	0 08	—	—	—
e hien	0 95	1 4	1 3	2 5
â ban	3 30	3 3	2 8	2 4
ê l'un	0 18	0 5	0 8	0 1
o bon	1 97	2 0	1 8	1 3
b lav	1 14	1 2	0 9	3 8
d clos	4 52	3 5	4 3	2 8
f fa	1 33	1 3	1 3	3 0
g guet	0 52	0 3	0 1	2 4
h car	3 81	4 5	3 7	5 2
l la	6 13	6 8	7 4	6 0
m na	3 17	3 1	3 1	4 5
n non	2 43	2 8	2 1	3 6
p pas	3 37	4 3	4 5	4 9
m rat	7 10	6 3	5 0	7 9
s sot	5 11	5 9	5 7	6 0
t toi	5 36	4 5	3 3	6 9
v vo	1 39	2 1	2 3	3 6
z azur	1 46	0 6	0 3	2 0
j chat	0 19	0 5	0 3	4 0
j joue	1 25	1 7	1 8	2 0
m oignon	0 12	0 1	0 0	1 4
y paille	0 23	0 5	0 2	1 2

ours et enfin, par les durées. Nous avons cependant cherché si ces déformations ne pouvaient avoir d'autres causes complémentaires plus générales que celles de la composition acoustique des phonèmes. Pour ceci nous avons utilisé plusieurs statistiques phonétiques.

1) D'abord, celle effectuée par P. CHASSAS sur des textes écrits anciens

et modernes. Cette statistique constitue une référence à la langue écrite notablement différente dans son utilisation de la langue parlée elle a donc un grand intérêt phonétique puisqu'elle représente une langue type fixée par les textes.

2) Nous en rapprocherons les statistiques effectuées sur des textes ayant servi à l'élaboration du Français Élémentaire : transcriptions de conversations qui nous ont été aimablement communiquées par G. Goughenheim, alors Directeur du Centre d'Etude du Français Élémentaire. Nous en avons tiré une statistique du français parlé notant la fréquence d'apparition des phonèmes sur 10 000 phonèmes explorés. Une deuxième statistique est en cours à partir des mêmes textes : elle porte sur les *transitions phonétiques* — est à dire sur les juxtapositions phonétiques dans la langue parlée (M. C. Richard). Nous utiliserons les résultats obtenus à partir de 7 500 transitions.

3) À titre de comparaison nous avons eu la curiosité de compter les phonèmes des mots les plus utilisés dans la langue parlée. Cette statistique inclut correspondant surtout aux formes grammaticales porte sur les 324 « formes les plus employées ». Elle répond à une langue qui serait réduit à ces 324 « mots » en tenant compte de la fréquence de leur utilisation (560 000 phonèmes sur 900 000 de l'ensemble des textes du Français Élémentaire).

4) Enfin nous avons recherché parmi tous les mots français de trois phonèmes quels étaient les phonèmes indispensables à l'identification du mot et nous avons calculé l'importance d'utilisation de chaque *forme phonétique caractéristique* (statistique inédite).

IV Comparaison des statistiques

1) Français écrit et parlé

Si l'on compare les statistiques du français écrit du français parlé et des formes les plus fréquentes on constate d'une façon générale que lorsqu'il existe une différence nette de pourcentage entre français parlé et écrit celle-ci est accentuée dans les formes les plus fréquentes (essentiellement grammaticales). Par exemple

CONSONNES (Pourcentage sur voyelles + consonnes)			
	Français écrit	Français parlé	Français « grammatical »
g	0.52	0.3	0.1
r	.40	.69	.50
h	5.36	.45	.33
z	1.46	0.6	0.7
l	6.43	.68	.74
p	3.37	.43	.45

VOYELLES (%)			
	Français écrit	Français parlé	Français « grammatical »
	.55	.65	.50
a	.536	.81	.84

	Français écrit	Français parlé	Français « grammatical »
è	3 01	5 3	5 9
ou	2 08	3 6	4 8
un	0 48	0 5	0 8

Ce dernier résultat tenant au fait que « un » est article et nombre, par conséquent très utilisé dans le français parlé. On remarque que pour « e » « u » l'évolution s'effectue vers l'ouverture de la voyelle donc vers plus d'intelligibilité. L'étude des transitions phonétiques montre en effet qu'un phonème ouvert augmente l'intelligibilité de la consonne qui précède par rapport au phonème fermé correspondant.

2) *Rôle informationnel*

Il semble que plus le phonème est utilisé dans des mots peu informationnels plus il perd ses propriétés de différenciation. Prenons les quatre phonèmes consonnantiques les plus fréquents dans les trois statistiques (« r », « l », « s », « t ») et comparons les à leur qualité d'opposition linguistique (phonème caractéristique). On constate que tout phonème devenu fréquent a peu de valeur relative d'opposition (« l » et « s ») et inversement (« r » et « t »).

CONSONNES (pourcentage entre consonnes)

	Français écrit	Français parlé	Français grammatical	Fonction d'opposition
r	14 8	14	10 8	10
l	12 8	13 8	16	8 3
s	11 2	11 6	12 4	8 3
t	10 6	9	4 6	9 6

Nous retrouvons dans ces résultats la grande loi d'équilibre linguistique : plus le phonème est utilisé en pratique courante, moins il est informationnel. Cette loi est également nette entre voyelle et consonne.

	Français écrit	Français parlé	Français grammatical	Fonction d'opposition
Voyelles (%)	49 6	49 1	53 4	28
Consonnes (%)	51 4	50 9	46 6	72

Il ne n'est pas valable pour les voyelles seules.

3) *Conditionnement phonétique*

Sur le plan de la pathologie, par contre, nous retrouvons les lois du conditionnement : plus le phonème est entendu, mieux ses caractéristiques sont fixées dans l'esprit du sujet.

On admet en général que dans les surdités les phonèmes aigus sont plus perturbés que les autres. Or, aussi bien pour les surdités que pour l'intégration, nous remarquons que chez l'enfant (« s » et « t ») phonèmes caracté-

ristiques rigues (« s » devant « t » ne descend pas en dessous de 4 000 c/s, une résonance caractéristique à 3 500) sont beaucoup mieux reconnus que d'autres fréquemment plus faciles à identifier (« s » 3 9% vient après « f » 14%, « v » 9 6%, « ch » 8 6%) (« t » 2 7% vient après « k » 7%, « p » 5 4%).

La reconnaissance au seuil donne entre « s » et « ch » une énorme différence d'intelligibilité (perturbation de 16 « s » lorsqu'un seul « ch » est mal reconnu) due uniquement à la composition acoustique de même entre « t » et « p ». Il y a donc pour l'enfant un autre facteur qui n'est pas acoustique c'est la répétition du phonème qui facilite l'apprentissage donc la reconnaissance.

On doit tenir compte dans les lois de l'intelligibilité de la *fréquence d'audition des phonèmes*. Cette règle est valable pour l'ensemble des phonèmes puis que chez l'enfant nous obtenons presque une statistique inversée. Les neuf consonnes les plus fréquentes représentent une moyenne de 3 9% des phonèmes perturbés les huit moins fréquentes une moyenne de 0 2%. Parmi les six consonnes les plus perturbées une seule se trouve dans les neuf plus fréquentes (« k »).

4) *Choix des mots*

Si le choix des mots peut être décidé par un effort conscient de l'individu si l'on choisit de préférence les mots le plus souvent entendus ce phénomène est beaucoup moins évident dans le choix des phonèmes. On doit admettre que le triage des mots fréquemment utilisés se fait en partie en fonction de leur contenu phonétique : lorsqu'on choisit entre deux mots synonymes on prend de préférence celui qui possède les phonèmes et surtout les transitions phonétiques les plus habituelles. Ce point est la pierre d'achoppement de l'utilisation des logatomes. Ceux-ci sont formés au hasard à partir des phonèmes d'une langue sans tenir compte de leur rentabilité ni surtout de leurs liaisons. Or beaucoup de liaisons phonétiques sont impropres à la langue ou très peu utilisées c'est une des raisons qui rend difficile l'utilisation des logatomes en audiométrie. Si l'on prend des mots usuels par contre en leur donnant par liste une improbabilité semblable à celle que l'on obtient avec les logatomes on peut alors effectuer une audiométrie indépendante du langage tout en gardant la structure acoustique habituelle.

V *La notion de phonème*

1) *L'acquisition des phonèmes*

Les définitions du phonème sont multiples suivant qu'elles sont données par le linguiste le psychologue ou l'acousticien. Lorsqu'on se penche sur les résultats des mesures phonétiques de l'audition on peut se demander jusqu'à quel point la notion d'une entité phonétique est valable en acoustique physiologique. Voici quelques faits.

a) Certains sourds entendent des phonèmes qu'étant donné leur seuil d'audition et la texture fréquentielle des phonèmes ils ne devraient pas reconnaître

L'intelligibilité des phonèmes est variable suivant la composition des mots nous avons noté l'influence des voyelles sur les consonnes tout se passe comme si la compréhension de la consonne dépendait de la voyelle dans une large part donc des transitions phonétiques

b) L'enfant n'a guère de notion spontanée du phonème en tant qu'entité. Il en prend conscience à l'apprentissage de la lecture, lorsqu'on lui montre un symbole graphique conventionnellement appelé à représenter un son déterminé. Et encore comme on le conditionne à juxtaposer ce son et sa transcription graphique il a du mal à concevoir qu'un groupe de deux ou trois lettres puisse ne représenter qu'un seul son phonétique. L'isolement du phonème conventionnel n'est donc pas spontané ni évident.

c) Il semble que l'enfant demi-sourd puisse nous apporter certains éléments : un mal entendant percevant le mot « asc » par exemple, reconnaîtra le « a » et le « é » phonèmes vocaliques sans difficultés acoustiques. Mais le « s » sera entendu comme s'il présentait une catástase « ■ s » suivie d'une tenue inaudible « s » puis d'une métastase « s-e » correspondant à la détente du « s ». Il reconnaît un début et une fin qu'il traduit par le phonème de type fréquentiel semblable avec une tenue silencieuse le « t ». L'enfant dira « até ». Ce qui permet d'identifier ce phonème correspond donc au passage voyelle-consonne et consonne-voyelle à défaut d'une tenue non perçue.

d) Dans l'apprentissage de la langue également le petit enfant commence par des lallations. Il prononce une mélodie qui a une signification dans son ensemble c'est un mot phonétique. Il reconnaît et apprend d'abord des variations sonores qui secondairement seront rattachées à des points d'articulation.

2) Les transitions phonétiques

Nous pensons donc que le passage d'un point d'articulation à l'autre représente la véritable expression phonétique, une statistique de transitions phonétiques complète très avantageusement la statistique des phonèmes isolés.

« a » est la voyelle la plus fréquente « r » la consonne la plus fréquente. Elles ont donc des chances d'être groupées. En effet la transition la plus fréquente est « a r » avec 1 04 %. Mais la transition « r-a » ne représente que 13 % venant en 36^e position. Voici par ordre de fréquence décroissante les premières transitions : « a r » (1 04) « p-a » (0 72) « i l » (0 62) « l a » (0 62) « r » (0 6) « a r » (0 6) « s i » (0 55) « w a » (0 55) « i l » (0 5) « on n » (0 2).

Nous n'avons aucune difficulté pour prononcer des syllabes construites suivant ces transitions. Pourtant on ne relève aucune systématisation des points d'articulations réciproques « r r » et « l l » ont des gestes articulatoires très différents. S'ils sont faciles c'est que nous avons continuellement l'occasion de les former ils peuvent être difficiles pour un étranger dont la langue

les exploite peu. Les particularités phonétiques des langues reposent selon toute vraisemblance beaucoup plus sur la fréquence des systèmes articulatoires que sur la nomenclature et la fréquence des phonèmes.

La connaissance du phonème et son identification dépendent souvent de ces transitions. Nous avons pu montrer qu'une explosive sourde suivant un « a » pouvait être identifiée sans qu'elle soit prononcée uniquement par la variation acoustique de la fin d'émission du « a ».

On peut d'ailleurs remarquer que les zones caractéristiques ne sont pas toujours atteintes dans un discours. L'expression acoustique essentielle est transitionnelle, se rapprochant plus ou moins des points d'articulation types. L'auditeur reconstitue à partir de ces variations le point de départ et la direction prise, extrapolant à un schéma acoustique que le sujet n'a pas besoin d'atteindre pour être intelligible.

3) Du phonème

Sur le plan linguistique, le phonème est une entité abstraite conventionnelle d'une structure acoustique définie dans ses rapports fréquentiels, non par des zones déterminées mais par opposition à d'autres phonèmes. C'est en quelque sorte les limites et non les centres qui donnent l'identité des unités phonologiques.

Sur le plan articulatoire, acoustique, psychologique, le phonème n'existe que sous forme de variations, c'est-à-dire d'opposition entre des timbres, opposition rendue sensible par un rapprochement progressif vers un timbre déterminé. Parmi ces variations acoustiques, le silence constitue lui-même une valeur phonétique d'opposition.

CONCLUSION

1) Dès que l'on essaie de fixer les valeurs linguistiques sous leur forme psychologique, on est tributaire des statistiques. Pour être valables, celles-ci doivent représenter une population suffisante, autant que possible homogène, et elles doivent répondre aux règles de la probabilité statistique.

Nous ne nous cachons pas que dans le travail présenté, les études expérimentales, tout particulièrement, ne sont pas suffisamment étendues pour refléter avec certitude des classifications entièrement valables. Nous les donnons comme une indication de l'ordre probable des perturbations phonétiques, nous réservant, par la poursuite de ces expérimentations, d'apporter ultérieurement des données vérifiées mathématiquement.

Le sens général de nos résultats reste valable, même si ultérieurement nous sommes amenés à reajuster les pourcentages donnés dans cette note.

2) Les différences phonétiques entre la surdité de l'enfant, souvent congénitale, et celle de l'adulte, en général acquise, marquent la place des suppléments possibles lorsque le phonème est déjà connu. Celles de l'enfant sourd et de l'enfant écoutant montrent le rôle de l'oreille dans le filtrage phonétique.

et permettent de comprendre ce qui sépare et ce qui relie l'hypocousie et le retard d'intégration

De même les lois générales de l'information et du conditionnement linguistique telles qu'elles apparaissent dans cette note démontrent des faits dont chacun perçoit intuitivement l'existence

Enfin l'interprétation que nous donnons de la notion du phonème et de sa place en physiologie en acoustique et en linguistique repose également sur des études d'analyses acoustiques qui n'ont pas été rapportées dans cette note. Elle s'intègre dans une conception psychophysologique de l'identification des signaux par opposition et apprentissage des critères d'opposition à partir de signaux variables et non de signaux stables

L'étude phonétique de l'audition pathologique est certainement un chapitre très important de l'audiologie clinique

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DISCUSSION

F. Escher Die interessanten Ausführungen von P. Mounier Kuhn sollten zu Untersuchungen mit der gleichen Methode in anderen Sprachen Anlass geben, da die Mischung von Vokalen und Konsonanten z. B. im Englisch und Deutsch sehr unterschiedlich vom Französisch ist. In der Schweiz hat das Problem grosse Bedeutung wegen der verschiedenen Dialekten.

P. Mounier Kuhn (Réponse) Je remercie le Prof. Escher de son intervention. L'étude présentée concernait seulement la langue française. Il est certain qu'il y aurait intérêt à faire des recherches analogues pour diverses langues et à en comparer les résultats. La question des transitions phonétiques mérite une attention toute particulière car elles sont un élément important de la structure de chaque langue.

THE EFFECT OF ULTRASONICS ON THE TEMPORAL BONE

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When applying ultrasonics to the labyrinth for destructive purposes it is necessary to observe the following points. (1) liquid coupling of the treatment rod to the bone must be provided. (2) the bone over the external semicircular canal must be flattened and reduced in thickness to $\frac{1}{2}$ mm. (3) the treatment rod and bone must be cooled to avoid thermal damage to the facial nerve; (4) the transducer must be maintained correctly in tune to avoid loss of ultrasonic output and the development of excessive side lobes which might endanger the facial nerve or cochlea.

INTRODUCTION

In spite of all the research that has been undertaken to study the effects of ultrasonic vibrations on animal tissues we are still very far from a full understanding of the changes that result from its application and of the processes by which these changes are produced. Arslan (1) showed that ultrasonics applied to the labyrinth can be successfully employed to destroy the vestibular end organ in the treatment of Meniere's disease. The investigations reported here have been performed with the ultrasonic generator which was designed and constructed by Arslan & Federici. One of these generators was acquired in 1957, but before using it clinically it was decided to investigate its physical properties and performance.

Description of the Apparatus

The transducer head of the Arslan Federici ultrasonic generator consists essentially of a cone to which is attached a disc shaped quartz crystal. This quartz crystal is mounted between two metal electrodes. When an alternating voltage is applied to these electrodes, the quartz contracts and expands in the same rhythm thus generating ultrasonic vibrations. In this apparatus the frequency of vibration is approximately one million cycles per second. These ultrasonic vibrations are then concentrated in the metal transformer cone and applicator rod, and issue as longitudinal vibrations from the tip of the rod when this is applied to a suitable medium. The lateral escape of undesired ultrasonics from the rod is prevented by a metal sleeve which surrounds the rod and is separated from it by a very narrow air gap.

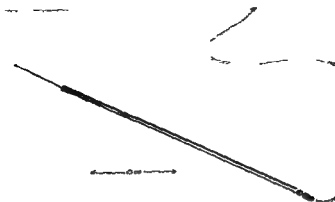


FIG. 1 Ultrasonic probe detector

Detection and Demonstration of Ultrasonics

At first attempts were made to map the intensities of the vibrations at various points in the ultrasonic beam in water with a small ultrasonic probe (Fig. 1). The sensitive tip of this probe consists of a barium titanate crystal 1 mm in diameter. The voltage generated by the crystal when exposed to ultrasonic vibration was indicated on a valve voltmeter. By this method it was possible to show that the ultrasonic beam of the Arslan transducer consists of a strong central ray together with a number of side lobes. A cross section of the beam is shown in Fig. 2. The intensity of the side lobes is not negligible, particularly

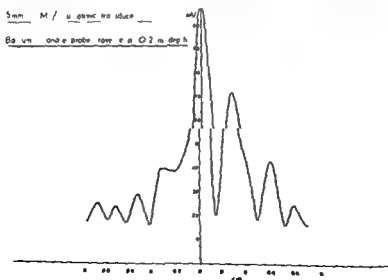


FIG. 2 Cross sections of ultrasonic beam measured with the ultrasonic probe

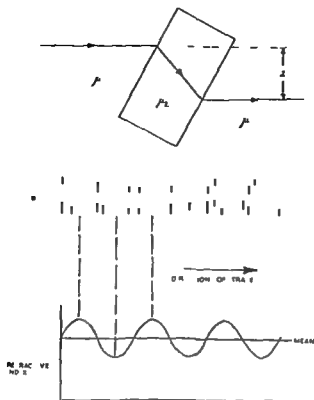


FIG 3 Basic principles of the Schlieren system

when the transducer drifts slightly from the correct tuning point with rising temperature. Owing to the standing waves set up by reflection of the ultrasonics from the detecting crystal surface different readings were obtained depending upon the exact angulation of the crystal in the ultrasonic field. This made the results of the measurements carried out with this method somewhat unreliable and it was therefore abandoned in favour of the Schlieren method which renders ultrasonic beams visible.

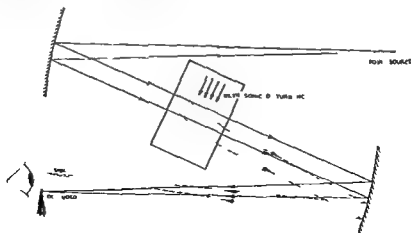


FIG 4 Diagram of the Schlieren system

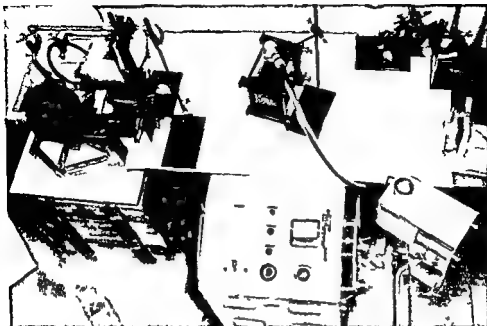


Fig. 1. Experimental arrangement of the Schlieren system.

The principle of the Schlieren method is shown in Fig. 1. A ray of light is refracted and therefore deviated laterally when it passes through a block of material whose refractive index differs from that of the surrounding medium. An ultrasonic beam consists of a rapid progression of compressions and rarefactions and therefore the refractive index of a medium traversed

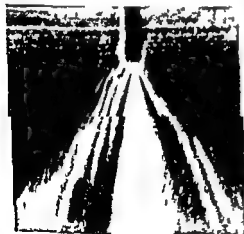


Fig. 1a

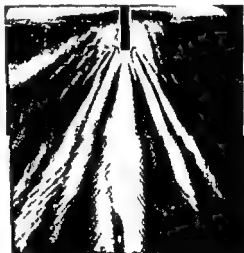


Fig. 1b

Fig. 1a. Schlieren photograph of the normal ultrasonic beam.

Fig. 1b. Schlieren photograph of the refraction from a transducer without an air gap sleeve. Reproduced by courtesy of the Journal of Laryngology.

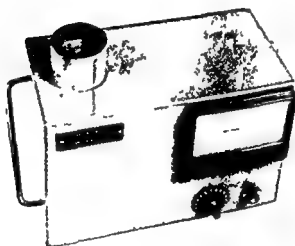
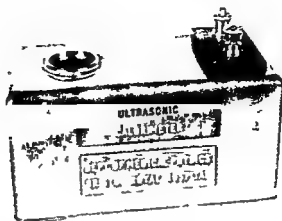


FIG. 8 Ultrasonic calorimeter

FIG. 9 Ultrasonic tuning monitor

by ultrasonics is constantly changing in the path of the beam. Fig. 4 shows diagrammatically the principle of the complete Schlieren system and Fig. 5 shows the experimental arrangement. Instead of the more usual lens system a reflective system using mirrors was chosen to avoid chromatic aberrations and will be published elsewhere (2). Figs. 6 and 7 are two examples of photographs which were obtained by this method. Fig. 6 shows a normal ultrasonic beam with the generator correctly tuned and Fig. 7 shows the great quantity of undesirable lateral radiation produced when the air gapped sleeve is removed.

The Measurement of Ultrasonics

As the meter on the control panel of the Arslan-Federici generator does not measure directly the ultrasonic output at the tip of the applicator rod,

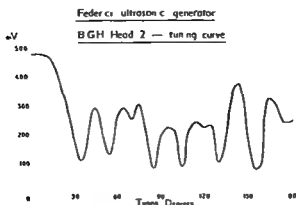


FIG 10 Tuning curve for the Federal ultrasonic generator Reproduced by courtesy of the Journal of Laryngology

a method had to be devised to do this. Although the ideal solution would be to monitor continuously the ultrasonic output during operation, there is at present no suitable method of achieving this although efforts are being made in this direction (3). Fig 8 shows the ultrasonic calorimeter we have developed for measuring the ultrasonic output of the generator. Details of its construction and use are given in a previous paper (4). Experiments carried out with this device have revealed that the tuning (and hence the ultrasonic output) varied considerably with changes of temperature resulting from warming up or change of ultrasonic power level. As this was very likely to occur during an operation, it was essential to devise a simple tuning monitor which could be used periodically to maintain the ultrasonic generator steadily in correct tune. Calorimetry is too slow for this purpose and the device which has been developed is illustrated in Fig 9. Fig 10 shows the output of this tuning monitor as the frequency control of the ultrasonic generator is swept over its full range. The correct peak (of the many alternatives) is that giving the best beam shape as shown on the Schlieren system, and the tuning monitor enables this setting to be steadily maintained. The practical importance of this proce-

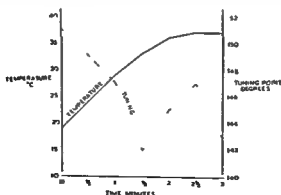


FIG 11 The variation of correct tuning point with applicator temperature and time



FIG 12



FIG 13

FIG 12 Schlieren photographs of well tuned and badly tuned ultrasonic beams

FIG 13 Schlieren photographs demonstrating the need for a liquid coupling film

ture is demonstrated by Fig 11 which shows the change of correct tuning point which occurs with change in cone temperature

Fig 12 shows the difference between a well tuned and a badly tuned beam. The method of tuning by observing the ultrasonic agitation of water in a Petri dish does not accurately correspond with the method just described. This is important because with correct tuning the ultrasonic beam is narrow and precise but in the wrong tuning position the central ray becomes wider and the side lobes relatively more intense. During application to the temporal bone these side lobes may endanger the facial nerve. It has been shown (3) that myelinated nerve fibres are one of the tissues most susceptible to the destructive effects of ultrasonics. It was this observation which led us to the conclusion that some of the cases of facial paralysis reported in the treatment of Meniere's disease might be due to excessively intense side lobes in a badly tuned ultrasonic beam.

Ultrasonic Coupling to the Temporal Bone

A factor of great practical importance concerns the coupling of the ultrasonic vibrations from the tip of the applicator rod to the temporal bone. The transmission of ultrasonic energy from a metal surface into air is negligibly

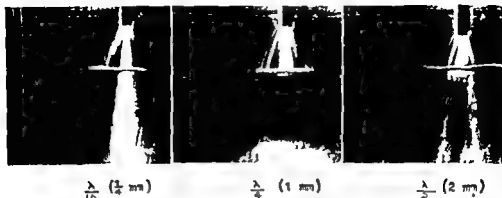


FIG 14 Schlieren photographs demonstrating the transmission of ultrasonics through bone laminae of various thicknesses. Reproduced by courtesy of the Journal of Laryngology

small. As technically it is impossible to produce a perfectly smooth flat surface to the temporal bone at operation, there must be small, irregular, air filled cavities between the applicator rod and the bone. This would inevitably mean poor and variable transference of ultrasonic energy. If the interface between rod and bone is filled with liquid, efficient transference of ultrasonic energy is achieved. Fig. 13 illustrates this point. In both pictures the ultrasonic generator is operating steadily into a bone slice mounted at the surface of a water tank. In the absence of coupling liquid the ultrasonic transmission is practically zero, whereas one drop of coupling water yields efficient transmission.

Ultrasonic Absorption and Interference

When ultrasonic vibrations pass from a medium of one acoustic impedance to a medium of another, some reflection, refraction and absorption of ultrasonics occur. During the passage of ultrasonics through any medium absorption takes place and this varies greatly with the acoustic properties of the medium and with the ultrasonic frequency. As the half intensity distance in temporal bone was found experimentally to be approximately $\frac{1}{2}$ mm at a frequency of 1 Mc/s, it was clear that in order to achieve maximum ultra-

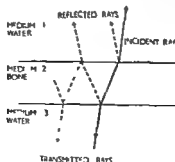


FIG 15 The principles of Interference

some energy transfer to the liquids of the labyrinth the bone overlying the perilymph must be pared down to the thinnest possible degree. Fig. 14 shows the passage of ultrasonics through different thicknesses of temporal bone in water. In the thickest slice (2 mm) a considerable transmitted intensity exists but there is appreciable attenuation. A 1 mm slice yields practically zero transmission while a $\frac{1}{2}$ mm slice gives practically complete transmission. Fig. 15 explains the complete arrest of the ultrasonic beam by a 1 mm bone slice. Part of the ultrasonic beam is reflected at the lower and upper bone faces in turn to emerge together with primary beam. With a bone thickness of 1 mm the extra path length of the twice reflected portion is 2 mm. This is exactly one half wavelength in bone at 1 mc/s resulting in 180° phase difference (and therefore cancellation) between the primary and reflected components. It is therefore very desirable to pare down the bone to a smaller thickness than this critical value. In practice thicknesses between $\frac{1}{2}$ and $\frac{3}{4}$ mm are achieved if the bone over the lateral semicircular canal is burred down until a clear blue line is visible.

Temperature Effects

When the very rapid absorption of 1 Mc/s ultrasonics in bone had been determined it was obvious that localized high temperatures might result from this absorption of ultrasonic energy and its transference into heat. Also due to the low overall efficiency of the present treatment head the applicator rod itself becomes hot its temperature varying with the ultrasonic power level. Thus two sources of bone heating exist namely the applied heat arising from the inefficient treatment head and the internal heating due to ultrasonic absorption. We therefore investigated (with a tiny thermocouple) the temperature effects at and near the tip of the applicator rod.

Fig. 16 shows the temperature rise at the bone surface directly beneath the rod when ultrasonics of varying intensity are applied. At 6 W/cm^2 the temperature rises to 90°C . Steady irrigation with saline at 37°C can reduce these

TABLE 1

Center level	Ultrasonic power (W/cm^2)	Transducer	Tip	Temperature ($^\circ \text{C}$)
		A	B	C
1	—	43	—	—
	9	51	—	—
4	33	61	11	11
7	9	90	16	44

A: No tip cooling. B: Tip cooled with 10 ml/min of water at 39°C . C: Tip cooled with 200 ml/min of water at 39°C .

FIG. 16. Table shows the relationship between applicator tip temperature, ultrasonic power and irrigation rate.

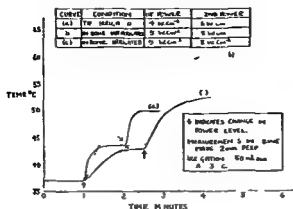


FIG 17 Surface and in bone temperatures with various power and irrigation conditions

temperatures to a safe level with modest flow rates as shown. A flow rate of 70 ml/min may be achieved with a normal constant level irrigator and a liquid head of approximately 1 metre.

Further experiments were performed to investigate temperatures reached at a point 2 mm deep in a temporal bone on the axis of the applicator rod. The results are shown in Fig 17 which contrasts conditions at the irrigated surface (curve a) with temperatures reached at a point 2 mm deep both with out (curve b) and with (curve c) irrigation at 50 ml/min. In each case a sudden change of ultrasonic power level has been made to demonstrate the rapidity of the resulting temperature rise. It is thought unwise to exceed a bone tem-



FIG 18 Schlieren photograph demonstrating how ultrasonics may be deflected round a curved channel



FIG. 19 Photograph, X-ray photograph and Schlieren photograph of a 2 mm thick slice of temporal bone in the vestibular region showing transmission of ultrasonics through an enlarged semi-circular canal.

perature of 44°C because of the close proximity of the facial nerve and possible thermal damage. Therefore, with the present apparatus ultrasonic powers exceeding 10 W/cm^2 are not employed at operation. This level is usually sufficient to ensure paralysis of the vestibular end organ in 15–25 min.

Further experiments are in progress to determine the relative importance of the two sources of bone heating previously referred to and will be reported fully in due course. They involve the construction of a non ultrasonic thermal replica of the applicator rod and comparisons of the temperature changes in bone produced by this device with those resulting from the present ultrasonic treatment head.

Already it has been found that more than 50% of the temperature rise 2 mm deep in bone is due to the applied heat. Therefore considerable benefit would result from the development of a more efficient ultrasonic treatment head in which this applied heat would be negligible. Work is proceeding in this direction.

Conduction in the Labyrinth

Since the temporal bone is such a poor conductor of ultrasonics it is unlikely that all parts of the labyrinth can receive an adequate dose for destructive purposes by this route alone. Schlieren photographs of the ultrasonic beam emerging from sections of temporal bone of different thicknesses (Fig. 14) illustrate this. The vibrations, however, are readily conducted by liquids. We consider that the main effect of ultrasonics on the neuro epithelium of the cristae and maculae is produced by vibrations transmitted through the fluids of the labyrinth and by shearing vibration resulting from the transformation of longitudinal to transverse vibrations at the bone liquid interfaces. The bony walls of the labyrinth being of very different acoustic impedance from the fluids probably act as reflectors on the same principle as a speaking tube causing reflection of the ultrasonics as they strike the wall. A Schlieren photograph (Fig. 18) shows the effect produced in a curved metal channel. On emerging from the end of the semicircular canal into the vestibule further complex reflections and interference patterns may develop we assume that the asymmetrical opening of the cochlea shields that organ from most of these vibrations. Photographs of the passage of ultrasonics through an enlarged external semicircular canal in a slice of temporal bone (Fig. 19) illustrate this effect.

ZUSAMMENFASSUNG

Die Wirkung der Ultraschallwellen an den Temporal Knochen

Die Untersuchungen über die hier berichtet wird wurden mit einem Arslan Federici Ultraschall Gerät ausgeführt. In dem medizinischen Schallkopf werden die Ultraschallwellen in einem Kegel konzentriert. Die unerwünschte seitliche Emission von Ultraschallwellen wird vermieden mit Hilfe eines Röhrchens das über den Stab des Schallkopfes gestulpt wird. Der innere Durchmesser des Röhrchens ist größer als der Durchmesser des Stabes, sodaß der Endstab des Schallkopfes mit einer Lufthülle umgeben ist.

Nachweis von Ultraschallwellen

In unseren ersten Experimenten versuchten wir das Feld des Ultraschallstrahles der von dem Ende des Schallkopfes ausgestrahlt wird mit Hilfe einer piezo elektrischen Ultraschallwellensonde auszumessen. Diese Methode wurde jedoch sehr bald aufgegeben da infolge der Reflexion der Schallwellen an der Oberfläche des Kristalls die Messungen zu ungenau waren. Wir gingen dann zu einem Schlierenverfahren über das unmittelbar das Bild der Ultraschallwelle liefert. Diese Methode beruht auf dem Prinzip daß der Lichtstrahl durch die Ultraschallwelle abgelenkt wird die den Lichtstrahl senkrecht durchkreuzt.

Messungen des Ultraschalls

Um die von dem Schallkopf ausgestrahlte Schallintensität zu messen wurde eine kalimetrische Methode benutzt. Im Verlaufe dieser Versuche wurde festgestellt

daß die Abstimmung sich mit Erhöhung der Temperatur verändert, die durch die Aufwärmung des Apparates verursacht wird. Da diese Erscheinung auch während einer Operation auftritt, muß die Abstimmung von Zeit zu Zeit kontrolliert werden. Für diesen Zweck wurde ein besonderer Abstimmungskontrollapparat konstruiert, mit dem die Abstimmung regelmäßig während der Operation kontrolliert wird. Eine gute Abstimmung ist auch aus einem weiteren Grund wichtig. Ein gut abgestimmter Strahl hat eine gute Richtcharakteristik, während ein schlecht abgestimmter Strahl eine Anzahl intensiver Nebenmaxima besitzt, die den Gesichtsnerven gefährlich werden können.

Flüssige und trockene Kopplung der Ultraschallwellen

In seinem ursprünglichen Artikel hat Prof. Arslan vorgeschlagen, daß die Berührungsstelle zwischen dem Ende des Stabes und des Knochens vollständig trocken gehalten wird. Wir haben gezeigt, daß die flüssige Kopplung einen größeren Wirkungsgrad hat als eine trockene Kopplung.

Absorption von Ultraschallwellen

Mit Hilfe der Schlieren-Methode kann gezeigt werden, daß die Ultraschallwellen im Knochen sehr schnell absorbiert werden. Die Intensität sinkt in einem $\frac{1}{2}$ mm zur Hälfte herab. Damit die Ultraschallwellen in das Labyrinth eindringen können, muß der darüber liegende Knochen zu einer möglichst dünnen Schicht heruntergeschliffen werden. Eine Schichtdicke von 1 mm muß um jeden Preis vermieden werden. Da diese Schichtdicke genau eine Viertel Wellenlänge darstellt, löschen sich der direkte Strahl und der zweifach reflektierte Strahl aus.

Thermische Wirkungen des Ultraschalls

Die Temperaturerhöhung an der Berührungsstelle ist mehr oder weniger proportional der Ultraschallwellenleistung, pro cm^2 . Wenn die Leistung C Watt/ cm^2 beträgt, Temperaturen können bis zu 90°C steigen.

Ultraschallleistung im Labyrinth

Die Schallintensität sinkt im Wasser in einer Entfernung von ungefähr 15 m zur Hälfte ab. Sobald die Ultraschallwellen die Flüssigkeit des Labyrinths erreichen, pflanzen sie sich durch wiederholte Reflektionen an den Wänden fort. Wir glauben, daß der Haupteffekt, der an dem Neuroepithelium des Labyrinths hervorgerufen wird, beruht auf der direkten Wirkung der Durchstrahlung der Labyrinthflüssigkeit mit Ultraschallwellen. Da wir eine flüssige Kopplung benutzen und die Schichtdicke des Knochens über dem Labyrinth soweit als möglich heruntergeschliffen wird, glauben wir, daß wir in der Hauptsache die direkten Wirkungen der Ultraschallwellen beobachten und daß die indirekten Wirkungen der Wärmeentwicklung ausgeschaltet sind. Die eine solche Gefahr für den Gesichtsnerv darstellen, da außerdem während der ganzen Operation Wasserkühlung angewendet wird.

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Unite Bristol Hospitals Bristol

DISCUSSION

Mr Arslan I want to express my great admiration to Dr Angell James for his experimental and clinical work concerning the ultrasonic destruction of the vestibular labyrinth in Ménière's disease. I am very glad to hear that Dr James has obtained very good results with this operation in his patients. I began to apply this method already in 1957. Since that year there have been operated in the University Department of Padua more than 600 cases of severe Ménière's disease (who did not receive the least benefit after all medical therapy). Moreover in the papers of Lumsden, Dubs, Altmann, Ariagno, Ironside and Lindsay and other authors there are reported the good results obtained by these authors with the same method.

The technical improvements and the refinement of the checking instruments of the Federici ultrasonic generator suggested and applied by Dr James and his co-workers are of extreme value as I could personally appreciate when I was present at an operation performed by him in his department at the Bristol Hospital.

I will adopt these modifications in my next surgical ultrasonic application.

L'ÉTUDE EXPERIMENTALE DU DÉPHASAGE DE L'ONDE ACOUSTIQUE LE LONG DE LA CHAÎNE OSSICULAIRE

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Le déphasage de l'onde le long de la chaîne des osselets est en rapport avec entre autres choses le facteur masse ossiculaire.

Il est intéressant, grâce au contrôle électrophysiologique d'étudier sur le chat la variation de la phase en fonction des variations de masse des osselets. Cette étude est notamment effectuée dans des situations comparables à celle créée chez l'homme lors de certaines tympanoplasties de type II et III et avec effet otolithaire composé.

Les résultats obtenus montrent des variations manifestes de la phase en fonction de la masse. Ils permettent dès maintenant d'orienter les actes chirurgicaux humains dans une certaine direction physiologique.

La physiologie de l'appareil tympano-ossiculaire fait intervenir la phase de la vibration stapedienne comme un des principaux éléments expliquant le mouvement compensé des deux fenêtres ovale et ronde, donc l'excitation du nerf cochléaire. Il est classique d'admettre que l'oscillateur ossiculaire retarde la phase de l'onde qui anime sa vibration. L'onde arrivant à la plume de l'étrier serait ainsi d'après certains auteurs en opposition de phase avec celle qui à travers le tympan et l'air de la caisse se présente à la fenêtre ronde. Il est en effet légitime de penser que la membrane élastique de la fenêtre ronde ne peut jouer pleinement son rôle compensateur que s'il y a opposition de phase entre fenêtres ovale et ronde.

Une confirmation indirecte de ce phénomène semble exister dans le fait suivant. Un porteur de perforation tympanique assez large peut garder une audition presque normale. La perte de l'effet protecteur du tympan vis-à-vis de la fenêtre ronde due à la perforation n'affecte pas l'audition si la chaîne ossiculaire continue à jouer son rôle, c'est-à-dire non seulement le maintien d'un certain rapport hydraulique (surfaces tympan-plume) mais aussi le déphasage du son par rapport à celui qui attaque directement la fenêtre ronde à travers la perforation.

On peut expliquer partiellement l'influence de la chaîne ossiculaire sur la phase par la valeur de sa masse. On sait que la masse d'un oscillateur retarde la phase tandis que sa rigidité l'avance (9). Devant l'importance que le physiologiste attache à cette influence de la masse ossiculaire sur le déphasage de l'onde acoustique et au rôle de ce dernier dans la stimulation cochléaire il nous a paru intéressant d'entreprendre une expérimentation sur le devenir

¹ Notamment dans les situations créées chirurgicalement chez l'homme.

de la phase dans des conditions tympano ossiculaires réalisées chirurgicalement chez l'homme

Il peut paraître en effet surprenant de constater des résultats auditifs souvent spectaculaires après des cicatrisations spontanées sur des tympanoplasties ayant supprimé le marteau et l'enclume, donc la masse ossiculaire (par exemple dans les tympanoplasties de type III Wullstein) : les succès ne seraient-ils pas le résultat d'une augmentation de la masse de la membrane vibrante tympanique elle-même ? et certains échecs ne trouveraient-ils pas une explication dans l'insuffisance de la masse de l'oscillateur tympanotrier créée par l'opération ?

Partant entre autres considérations de ce principe physiologique Clavier (6) proposa dans notre Clinique d'utiliser chaque fois que cela est possible la tête du marteau par rotation autour de son tendon musculaire pour l'interposer entre tête de l'otrier et membrane vibrante tympanique le maintien de la plus grande partie du marteau dans cette position donnant à l'oscillateur de l'oreille moyenne une certaine masse

Moyens expérimentaux

I I animal

Le chat fut choisi comme animal d'expérience. Sa chaîne ossiculaire est parfaitement accessible sous microscope par atticotomie. Une trepanation plus inférieure permet de placer une électrode sur la fenêtre ronde.

II I intervention

L'animal est endormi à l'aide de nembutal puis trachéotomisé et curarisé. Dès l'effet de ce dernier il est placé en respiration artificielle sur pompe Baudouin.

L'opération comprend ensuite les temps suivants :

— incision postérieure : dégagement de la bulle ouverte à la fraise dans l'axe de la fenêtre ronde : mise en place de l'électrode sur cette dernière : fermeture de la trepanation avec du ciment dentaire

— puis incision postéro-supérieure : ouverture de l'attique afin de mettre en évidence la chaîne ossiculaire sans la léser : fermeture hermétique et provisoire de cette trepanation par un fragment de papier humecté ou d'une feuille de matière plastique

— ablation du pavillon et fixation dans le conduit d'un tube de polyéthylène afin d'amener la stimulation sonore directement. L'animal est ensuite placé dans la chambre de silence : les autres opérations chirurgicales y seront effectuées.

Une électrode indifférente est placée à la plaie : le sujet est relié également à la terre.

III I appareillage

1^{er} Principe. Pour mesurer les écarts de phase de la vibration acoustique au niveau de l'organe récepteur de Corti nous avons estimé nécessaire d'utiliser la méthode suivante :

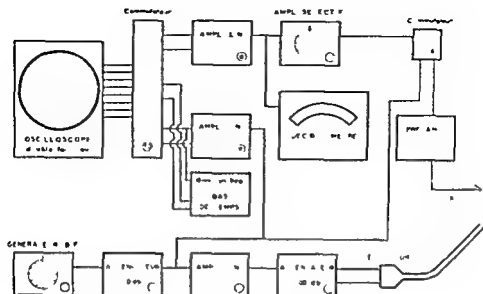


Fig. 1. Schéma de l'appareillage.

a) Stimulation par une vibration sonore sinusoïdale pure de fréquence déterminée

b) Recueil du potentiel microphonique cochléaire en un point fixe (toujours le même) à la fois au cours d'une expérimentation sur un même animal — grâce à une électrode fixe à demeure par du ciment dentaire — ainsi que chez les différents animaux ayant servi à notre étude: le point de recueil utilisé est la fenêtre ronde.

c) Comparaison de la phase entre les deux potentiels électriques suivants enregistrés sur l'écran d'un oscilloscope cathodique à double trace: potentiel sinusoïdal appliqué dans l'écouteur stimulant l'oreille du sujet et potentiel de même fréquence recueilli par l'électrode.

2° Les appareils. Ceux-ci peuvent être présentés par leurs deux fonctions distinctes bien que les deux parties affectées à ces deux fonctions forment un tout qui a été spécialement assemblé — pour fonctionner de pair — en vue de l'étude des phénomènes de déphasage des courants.

Nous distinguerons cependant pour la clarté de l'exposé le système de stimulation acoustique et le système d'enregistrement des potentiels cochléaires.

A. Système sonore stimuloire. Il se compose des appareils suivants (voir fig. 1).

1. Un générateur de fréquences sinusoïdales (n° 1) CRC type BF GB 62 capable de délivrer toutes les fréquences de 30 à 300 000 Hz par réglage continu.

Pour ces recherches nous nous sommes limités aux fréquences de 500 1 000 2 000 10 000 Hz. La tension de sortie est réglable entre 0 et 10 Veff de manière continue. Il est possible d'ajuster le seuil d'audition (voir au paragraphe « atténuateurs »).

Un amplificateur (n° 3) de puissance à forte contre-réaction linéaire de 20 à 40 000 Hz n'introduisant qu'un taux de distorsion minimale gain fixe 60. Sortie push pull directe. Puissance modulée 20 Watts.

■ Deux atténuateurs, gradués en décibels¹ L'un (n° 4) de 10 en 10 db, couvre une étendue de 100 db L'autre (n° 90) de 2 en 2 db, couvre une étendue de 0 à 10 db Tout l'intervalle de 0 (seuil) à 110 db est donc couvert de ■ en 2 db Le seuil physiologique d'audition est ajusté par le réglage du niveau de sortie du générateur (B F n° 1)

4 Un écouteur Socatex, type SP6 Celui-ci est inclus dans un bloc de plexiglass épais, ayant un espace intérieur conique dont la base est la membrane de l'écouteur et dont le sommet est percé, ce qui constitue la seule communication extérieure Cette ouverture est branchée hermétiquement et directement sur un tube de polyéthylène de très fin diamètre intérieur (1 mm) de façon à limiter au maximum les fuites sonores Le tout est enveloppé dans une boîte à double paroi étanche de bois et de cuivre, dont les espaces intérieurs libres sont garnis d'ouate de verre Ceci assure à la fois une isolation phonique satisfaisante et un isolement électrique parfait (aucune action sur l'appareil récepteur, à pleines puissances stimulateur et d'amplification n'a pu être décelée)

Le tuyau de sortie est mis directement en relation avec le conduit auditif du sujet, à l'intérieur duquel il est suturé Un raccord amovible placé sur le parcours du tuyau permet de séparer l'écouteur de l'animal, sans avoir besoin de couper les points qui fixent le tuyau de façon à effectuer plus commodément les interventions chirurgicales

B Système d'enregistrement des potentiels cochléaires Il se compose des appareils suivants (voir fig. 1)

1 L'électrode celle-ci est appliquée sur la fenêtre ronde Elle est solidement fixée sur la paroi osseuse de la caisse par du ciment dentaire Une fois mise en place, on ne modifie rien à sa position jusqu'à la fin de l'expérience Cette électrode est un fil d'argent, diamètre 0.08 mm recouverte de vernis isolant, dénudée sur l'extrémité destinée à être appliquée sur la fenêtre ronde, c'est à dire sur une longueur totale d'environ 1 mm cette partie non isolée est pliée en forme d'anneau de façon à reposer à plat sur la membrane sans la perforer Cette manière de procéder présente en outre les avantages suivants : faible pression de contact nécessaire ne perturbant donc pas la réception normale du son appliqué à l'oreille, élasticité de l'extrémité pliée du fil assurant un contact toujours satisfaisant avec le liquide imbibant la membrane, quelle que soit la pression intra-labyrinthique résistance de contact très faible, réduite à une valeur inférieure à 25 k Ω , ce qui facilite grandement l'enregistrement des potentiels, qui sont, en effet recueillis par la méthode monopolaire, d'où la nécessité de ne opérer qu'à des impédances d'électrode les plus basses possibles

2 Un pré-amplificateur (n° 5) linéaire de 20 à 30000 Hz alimentation continue, gain fixe 15000

3 Un post-amplificateur (n° 7) — destiné à alimenter directement les plaques défectrices de l'oscilloscope cathodique — linéaire de 20 à 30000 Hz, gain réglable de 0 à 400

4 Un amplificateur sélectif A O I P, type L L 40 Cet appareil est un amplificateur à contre réaction sélective cette dernière étant appliquée par l'intermédiaire d'un filtre suppresseur de bande en double T, du type « résistance-capacité » dont la

¹ Ces atténuateurs sont intercalés dans le circuit de la façon suivante Le plus réduit (domaine de 10 db) avant l'entrée de l'amplificateur comme l'indique le schéma de la fig. 1 (n° 2) L'autre (longue de 100 db) entre le 1^{er} et le 2^e étage de l'amplificateur de puissance Pour simplifier l'aspect du schéma ce dernier indique son emplacement juste avant l'écouteur (n° 4) L'effet résultant sur l'intensité des stimulus sonores doit être considéré comme négligeable

fréquence utile peut être réglée de manière continue, à volonté de 12 à 12000 Hz. Ce système « amplificateur + contre réaction sélective » est équivalent à un filtre « passe bande » jouissant en outre de deux avantages : fréquence réglable de façon continue entre 12 et 12000 Hz, et gain supérieur à l'unité (amplification).

L'atténuation est de 35 db sur les harmoniques 2 de part et d'autre de la fréquence choisie. Cette atténuation permet d'obtenir une excellente stabilité de l'enregistrement permettant de réaliser des photographies de l'écran très nettes et qui améliore la précision des mesures.

5 Un oscilloscope cathodique à double faisceau, à véritable balayage simultané, équipé d'un tube DuMont, à écran bleu pour photographie.

6 La base de temps est d'un type classique. Le « balayage » est synchronisé sur le potentiel de sortie du générateur B1.

7 Une caméra Cossor, qui fut utilisée « par vues séparées ». Aucune mesure n'est effectuée en cours d'expérimentation. Seules sont prises des séries de clichés pour chaque fréquence choisie. Les mesures se font ensuite sur les projections d'images agrandies par un « lecteur » pour microfilms.

8 Un millivoltmètre-décibelmètre électronique est utilisé, de plus pour mesurer l'amplitude de la réponse. Cette étude des amplitudes fait l'objet d'une autre série d'études dont certaines parties ont été déjà publiées (3). L'appareil est branché en dérivation sur la sortie de l'amplificateur sélectif.

9 Les commutateurs (n° 6 et n° 10). Le commutateur n° 6 permet de pratiquer soit l'accord des appareils sur la fréquence choisie, soit l'enregistrement des réponses de l'animal. Le commutateur n° 10 permet d'obtenir l'action des différents potentiels sur différentes plaques de déflexion du tube de l'oscilloscope en fonction des nécessités de l'expérimentation. Il en sera parlé « Mesures des différences de phases » (voir à ce paragraphe).

L'expérimentation et résultats

L'expérimentation a essentiellement porté sur l'étude de la phase et non de l'intensité de la réponse microphonique.

Six chats furent mis en expérimentation. Les interventions n'ayant pas donné toutes garanties de perfection sur les deux premiers, seuls les résultats de quatre d'entre eux seront mentionnés ici.

1 Mesure des différences de phases obtenues

Pour chaque situation expérimentale, trois fréquences furent étudiées : 500, 1000 et 10000. Les intensités étaient choisies de sorte que l'amplitude de la réponse soit comparable et bien visible. Le problème de l'intensité sera analysé dans la discussion.

Les mesures sont effectuées grâce aux films d'enregistrement. Les enregistrements sont de deux sortes :

1° Deux tracés sinusoïdaux : tracé inférieur - potentiel stimulant les écouteurs, tracé supérieur - potentiel recueilli sur l'animal et amplifié.

Le balayage des deux spots (tant simultané) il suffit de mesurer le décalage horizontal des deux sinusoïdes. Pour cela, les commutateurs n° 6 et 10 réalisent les connexions du genre α (voir tableau 1).

2° Tracés d'essai : l'envoi simultané du potentiel stimulant les écouteurs et du potentiel recueilli sur l'animal et amplifié, chacun sur une des deux paires de plaques de déflexion influençant un même spot, impose à ce dernier un déplacement elliptique.

TABLEAU 1 Tableau d'emploi des commutateurs II et I de la figure 1

Ce tableau indique les organes des appareils auxquels sont reliés les appareils désignés dans le haut des colonnes : Les paires de plaques de l'oscilloscope donnant une déflexion dans le sens vertical sont notées $\vee \vee$ Celles donnant une déflexion dans le sens horizontal sont notées $\wedge \wedge$ Indice 1 pour le faisceau inférieur indice 2 pour le faisceau supérieur Ex $\wedge 2 \wedge 2$ = déflexion horizontale du tracé supérieur

Communication Genre	Commutateur n° 6	Commutateur n° 10 (voir schéma fig 1)		
	Amplificateur sélectif	« Base de temps »	Amplifi- cateur n° 8	Amplifi- cateur n° 9
α	Pré amplificateur n° 3	$\wedge 1 \wedge 1$ $\wedge 2 \wedge 2$	$\vee \vee$	$\vee 1 \vee 1$
β	Pré amplificateur n° 3	\wedge éant	$\vee \vee$	$\wedge \vee$
α	Générateur B F	\wedge éant	$\vee \vee$	$\wedge \vee$

tique l'excentricité et l'inclinaison de l'ellipse permettent aussi une mesure du déphasage des deux courants à comparer Pour cela les commutateurs n° 6 et 10 réalisent les connections du genre β (voir tableau 1)

La mesure du déphasage ainsi réalisée n'est pas en elle même une « valeur absolue » car il faut tenir compte de déphasages supplémentaires introduits par l'écouteur, par le trajet écouteur tympan et dans la chaîne d'amplification Ce dernier est cependant très faible inférieur à 5°

Cette mesure a une valeur comparative d'un état de l'animal à un autre, parce que les effets ci dessus se reproduisent toujours identiques à eux mêmes pour une même fréquence donnée et n'interviennent donc pas dans la différence mesurée lors de deux états de l'oreille, pour une fréquence égale dans ces deux états

Si nous appelons φ la phase du potentiel recueilli sur l'animal normal à oreille intacte et φ_1 la phase du potentiel recueilli sur l'animal ayant subi un premier type d'intervention chirurgicale l'écart de phase (notation $\Delta_1 \varphi$) introduit par l'intervention réalisée sera

$$\Delta_1 \varphi = \varphi_1 - \varphi_0$$

φ_1 et φ_0 étant exprimés comme les écarts de phase du potentiel de la réponse de l'animal par rapport au potentiel appliqué aux écouteurs qui a pour convention une phase prise égale à zéro

Dans nos résultats nous n'avons pas mentionné φ_0 , φ_1 , φ_2 qui sont sans intérêt réel mais uniquement les $\Delta_1 \varphi$, $\Delta_2 \varphi$ qui sont seuls intéressants étant entendu qu'alors la convention adoptée veut que $\Delta_1 \varphi = 0$

II Différentes situations de l'appareil tympano ossiculaire analysées (voir fig 3)

Les différentes opérations furent les suivantes

A Recherche de la phase à l'état normal Cette donnée n'a aucune valeur absolue puisqu'elle dépend non seulement de l'animal mais de l'appareillage Pour chaque animal elle représente le « zéro de base » par rapport auquel sont calculées toutes les valeurs des phases obtenues selon les différentes modalités expérimentales

fréquence utile peut être réglée de manière continue, à volonté de 12 à 12000 Hz. Ce système « amplificateur + contre réaction sélective » est équivalent à un filtre « passe bande » jouissant en outre de deux avantages : fréquence réglable de façon continue entre 12 et 12000 Hz et gain supérieur à l'unité (amplification).

L'atténuation est de 15 db sur les harmoniques 2 de part et d'autre de la fréquence choisie. Cette atténuation permet d'obtenir une excellente stabilité de l'enregistrement permettant de réaliser des photographies de l'écran très nettes ce qui améliore la précision des mesures.

5 Un oscilloscope cathodique à double faisceau, à véritable balayage simultané équipé d'un tube DuMont, à écran bleu pour photographie.

6 La base de temps est d'un type classique. Le « balayage » est synchronisé sur le potentiel de sortie du générateur H.F.

7 Une caméra Cossor, qui fut utilisée « par vues séparées ». Aucune mesure n'est effectuée en cours d'expérimentation. Seules sont prises des séries de clichés pour chaque fréquence choisie. Les mesures se font ensuite sur les projections d'images agrandies par un « lecteur » pour microfilms.

8 Un millivoltmètre déshelmètre électronique est utilisé, de plus pour mesurer l'amplitude de la réponse. Cette étude des amplitudes fait l'objet d'une autre série d'études dont certaines parties ont été déjà publiées (1). L'appareil est branché en dérivation sur la sortie de l'amplificateur sélectif.

9 Les commutateurs (n° 6 et n° 10). Le commutateur n° 6 permet de pratiquer soit l'accord des appareils sur la fréquence choisie, soit l'enregistrement des réponses de l'animal. Le commutateur n° 10 permet d'obtenir l'action des différents potentiels sur différentes plaques de déflexion du tube de l'oscilloscope en fonction des nécessités de l'expérimentation. Il en sera reparlé « Mesures des différences de phases » (voir le paragraphe).

L'expérimentation et résultats

L'expérimentation a essentiellement porté sur l'étude de la phase et non de l'intensité de la réponse microphonique.

Six chats furent mis en expérimentation les interventions n'ayant pas donné toutes les garanties de perfection sur les deux premiers, seuls les résultats du quatre d'entre eux seront mentionnés ici.

1 Mesure des différences de phases obtenues

Pour chaque situation expérimentale trois fréquences furent étudiées 500 1000 et 4000. Les intensités étaient choisies de sorte que l'amplitude de la réponse soit comparable et bien visible. Le problème de l'intensité sera analysé dans la discussion.

Les mesures ont été effectuées grâce aux films d'enregistrement. Les enregistrements sont de deux sortes.

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Le balayage des deux spots étant simultané il suffit de mesurer le décalage horizontal d's deux sinusoïdes. Pour cela les commutateurs n° 6 et 10 réalisent les connexions du genre α (voir tableau 1).

2° Tracés du 1 et du 2. L'envoi simultané du potentiel stimulant les écouteurs et du potentiel recueilli sur l'animal et amplifié - chacun sur une des deux paires de plaques de déflexion influençant un même spot - impose à ce dernier un déplacement elliptique.

TABLEAU 2 Tableau des modifications de phases obtenues sur quatre animaux pour trois fréquences 500, 1000 et 4000 et dans six conditions expérimentales différentes B, C, D, E, F, et G par rapport à la normale A

Les cases vides correspondent à des situations expérimentales qui n'ont pu pour des raisons diverses être réalisées sur les animaux correspondants

Expériences références	Expérience n° 3 (600 420 A 321 A)			Expérience n° 4 (600 423 A 321 A)			Expérience n° 5 (600 430 A 321 A)			Expérience n° 6 (600 528 A 321 A)		
	500	1000	4000	500	1000	4000	500	1000	4000	500	1000	4000
Normal	0	0	0	0	0	0	0	0	0	0	0	0
Ablation de l'enclume	-19	-20	+8	-33	-88	-13	+43	-93	-136	+206	+198	+36
										+158	+212	+51
Tympanoplastie con- tact type Glavert				+138	+73	-104				+74	+64	+22
Tympanoplastie type III tête marteau enlevée	+168 ±180	+137 +102	+86 +80 ^a	113 -167	-147 ±180	-113 -100 ^a	+24	+103	±180	-122	-106	±180
Interposition libre de tête du marteau	+123	+121	+135				+34	112	±180			
Interposition libre enclume	29	+13	+31	+22	+120	-97						
Interposition libre enclume + tête du marteau	+81	+133	+172	64	61	-96	+34	+12	-180			

^a La deuxième ligne représente les résultats obtenus lors d'un contrôle final après toutes les autres opérations. Elle montre des chiffres voisins de ceux de la première ligne correspondant à la réalisation du type III en début d'expérience. Ainsi à plusieurs heures d'intervalle et après différentes manipulations la même situation donne sur le même animal le même résultat.

enlevée est interposée librement entre l'imbeau tympano mental et tête de l'etrier.

F Contact entre l'imbeau tympano mental et etrier avec interposition entre les deux de l'enclume libre. L'enclume préalablement enlevée est interposée librement entre l'imbeau tympano mental et etrier.

G Contact entre l'imbeau tympano mental et etrier avec interposition entre les deux de l'enclume et de la tête du marteau. L'enclume et la tête du marteau préalablement enlevées sont interposées librement entre l'imbeau tympano mental et etrier.

III Résultats

Les résultats des différentes opérations sont consignés dans le tableau 2.

Mise à part l'ablation de l'enclume (B) qui soulève des problèmes particuliers du fait de l'absence d'un « effet columellaire » les commentateurs peuvent raisonnablement porter sur les autres expériences

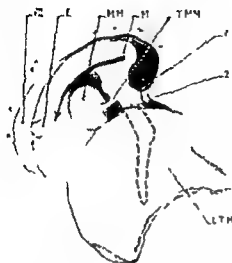


Fig. 2

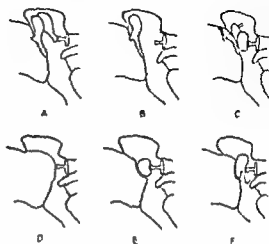


Fig. 3

Fig. 2 Réalisation d'un « effet columellaire composé » chez le chat par fixation de la tête du marteau sur la tête de l'étrier autour de l'axe tendineux du tenseur tympani (tempo opératoire « C » de notre expérimentation). L T M lambeau tympano-métal M tête du marteau M M muscle du marteau (tensor tympani) T M M tension du muscle du marteau (seule attache non sectionnée) L étrier A H canal de Hall per 1 section de l'apophyse antérieure du marteau 2 section du col du marteau

Fig. 3 Différentes situations expérimentées. A situation normale B ablation de l'enclume sans recollage du conduit et sans étrier C effet columellaire composé (type I laurier) rotation de la tête du marteau et interposition de celle-ci entre membranes vibrantes et tête de l'étrier D situation de la tympanoplastie type III de Wullstein E la tympanoplastie de la tête du marteau sans insertion de la membrane vibrante et tête de l'étrier F interposition de l'enclume entre membranes vibrantes et tête de l'étrier

B *Ablation de l'enclume* Cette opération provoque une surdité de transmission importante de 50 à 60 db (environ (5)) de ce fait la voie osseuse doit intervenir sur la phase de l'onde microphonique il faut en tenir compte lors de l'interprétation des résultats point qui sera évoqué à la discussion

C *Tympanoplastie avec effet columellaire composé type I laurier* (voir fig. 4) Une tympanoplastie est alors effectuée après ablation de l'enclume par recollage du lambeau tympano-métal, élargissement du conduit osseux à la fraise afin de voir l'étrier, section de la tête et de l'apophyse antérieure du marteau, rotation de la tête du marteau sur son axe tendineux et application sur la tête de l'étrier, application hermétique (autant que l'on se peut) du lambeau tympano-métal sur la tête du marteau et le pourtour de l'attache columelle.

D *Tympanoplastie type III Wullstein classique* Ablation de la tête du marteau et application hermétique du lambeau tympano-métal directement sur la tête de l'étrier. La masse ossiculaire a donc pratiquement disparu.

E *Contact entre lambeau tympano-métal et étrier avec interposition entre les deux de la tête libre du marteau* La tête du marteau qui fut préalablement

c) *L'essai de rétablissement de la masse ossiculaire par l'interposition libre des osselets eux mêmes entre membrane vibrante et étrier* (E, F et G) ne donne pas constamment de résultats probants dans nos conditions d'expérience. Sauf pour certains cas, le manque d'adhérence des fragments ossiculaires empêche probablement que leur masse puisse régulièrement intervenir sur la phase de la vibration du système tympano-étrier. Ceci explique la diversité des résultats obtenus dans les situations L, F et G.

Lorsque l'osselet peut être bien interposé, par exemple l'enclume de la série I lors de l'expérience n° 3 (l'animal n° 600 420 A-321-A) sa masse provoque incontestablement un retour de la phase au voisinage de la normale (voir tableau 2 et fig. 5). Si, au contraire, la forme de l'osselet (rondité de la tête du marteau) (I) ou l'insubilité de « l'effaillage » des deux fragments (enclume et tête du marteau) (G) compromet l'adhérence au système oscillant, les résultats restent voisins de ceux obtenus sans l'addition de ces osselets libres. C'est à dire dans la situation d'une tympanoplastie type III (D).

DISCUSSION

De ces expériences, il ressort que la suppression de la masse ossiculaire même si il existe un bon contact (effet columellaire) entre la membrane tympano-membrane et l'étrier (cas d'une tympanoplastie de type III) entraîne une modification importante de la phase, se rapprochant plus ou moins de l'opposition de phase. Si l'on arrive à maintenir entre autres facteurs une certaine masse ossiculaire, ce déphasage est beaucoup moins important. Avant de discuter la valeur de ces résultats sur le plan de la chirurgie humaine, il est nécessaire de soulever les critiques de la méthode expérimentale utilisée.

1° Critiques de la méthode expérimentale utilisée

Plusieurs problèmes peuvent être envisagés :

a) Influence des muscles de l'oreille moyenne

En augmentant la rigidité de la chaîne des osselets, la contraction musculaire du *stapedius* et du *tensor* peut modifier considérablement les caractéristiques du système oscillant tympano-ossiculaire. C'est pour supprimer cette cause de variation dans les réponses que toutes ces expériences furent menées sous curarisation profonde avec respiration artificielle. On peut cependant supposer qu'une certaine tonicité musculaire aurait pu réapparaître et modifier quelques réponses à la fin du laps de temps correspondant à l'effet de la précédente injection curarisante. C'est la raison pour laquelle la curarisation fut renouée à la demande et les expériences effectuées immédiatement après l'apparition de la résolution musculaire complète et la mise en circuit de la pompe respiratoire.

b) Influence de la voie osseuse

Lorsque l'appareil de transmission est endommagé profondément la stimulation par voie acoustique peut atteindre la cochlée en provoquant la vibration de l'os temporal à condition que son intensité soit suffisamment élevée (au moins 30 à 40 db). Or pour obtenir des réponses bien photographiables notamment pour les figures de l'isogyne des intensités supérieures à 40 db ont souvent été nécessaires dans nos expériences. Une stimulation osseuse seule ou surajoutée à la stimulation cheminant à travers l'appareil tympanique était alors certaine et risquait de modifier la phase des réponses obtenues et leur interprétation. Il paraît donc nécessaire d'analyser ce point particulier pour dissiper cette incertitude.

On peut considérer la vibration osseuse obtenue lors d'une stimulation acoustique dans deux circonstances différentes : en cas de surdité de transmission profonde par manque d'effet columellaire et en cas de surdité de transmission incomplète car l'appareil de transmission fonctionne du fait que l'effet columellaire a pu être respecté.

1. *Suppression de l'effet columellaire* L'ablation de l'enclume (expérience B) sans rétablissement du contact columellaire détermine une telle situation la stimulation pour provoquer des réponses variables est au dessus de 30 à 60 db et se transmet essentiellement par voie osseuse.

Il est à peu près certain qu'au dessous de 2 000 Hertz l'audition par voie osseuse obéit au même « d'inertie » (1) notamment « inertie » des structures de l'oreille moyenne (comme l'a démontré récemment J. P. Legoux la masse et la rigidité ossiculaires influencent alors directement l'amplitude et la phase de la réponse microphonique à une stimulation transosseuse (2).

L'ablation de l'enclume (expérience B) pouvait donc à première vue avoir un intérêt dans notre travail pour analyser l'influence de la seule présence de l'étrier sur la phase. Seule la masse stapédienne est alors en cause puisque l'étrier est libre du reste du système tympano-ossiculaire et pour faire image « attiré(e) en quelque sorte de dedans en dehors ». Cependant cette situation n'est jamais recherchée chirurgicalement chez l'homme puisque en l'absence de contact columellaire elle est responsable de surdité profonde. Elle ne peut donc être utilisée dans ce travail centré sur des situations créées par la chirurgie humaine et axé sur une stimulation « de dehors en dedans » à partir de celle de la membrane tympanique elle-même.

Deux sortes de stimulation aussi dissimilaires ne peuvent donner de réponses comparativement utilisables dans la même expérimentation trop de facteurs différents pouvant intervenir c'est la raison pour laquelle nous n'avons pas cherché à interpréter les résultats de l'expérience B. Par contre ce genre d'expérience sans effet columellaire peut s'inscrire dans le cadre de recherches portant sur la voie osseuse elle-même et les mécanismes de l'inter-diffusion telles celles déjà effectuées par d'autres auteurs (2, 3, 6, 7 et 8).

2. *Conservation de l'effet columellaire* Dans les autres expériences avec effet columellaire (C, D, L, I, G) il a parfois été nécessaire de stimuler à des intensités supérieures à 40 db pour obtenir de belles figures de l'isogyne.

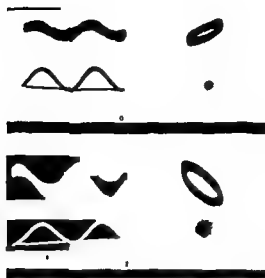


Fig. 7) Dépharyngé provoqué par la présence de sang sur le tympan (animal N° 600398)

fig. 7) Or, malgré le soin apporté à l'expérimentation, il n'est pas possible de nier après un laps de temps plus ou moins long l'apparition de sécrétions ou de sang ayant pu modifier les réponses. Par contre, si le sang s'accumule en faible quantité dans la bulle tympanique son aspiration ne modifie pas les réponses obtenues.

2° *Interprétation de ces résultats expérimentaux sur le plan de la chirurgie humaine*

La transposition de nos résultats sur le plan de la chirurgie humaine ne peut se faire sans réserve. Sur le plan physiologique, les chirurgiens ont parfois une certaine répugnance à accepter d'emblée des données vérifiées uniquement sur l'animal. Il est certain qu'on trouve des différences de détails entre l'oreille moyenne du chat et celle de l'homme; cependant on peut admettre que s'il existe des variations quantitatives dans les réponses fournies par les expériences portant sur différents mammifères (y compris l'homme) il y a toujours similitude qualitative, ainsi que du sens des réponses. Sur le plan physiologique général, nos résultats nous paraissent donc parfaitement transposables.

En ce qui concerne le point plus particulier des conséquences de l'acte chirurgical lui-même, les conditions de vérification expérimentales sont très éloignées de celles de la chirurgie humaine. Sur l'animal, des contrôles microphoniques sont effectués immédiatement après l'intervention; les différents éléments du système oscillant que l'on a cherchés à créer ne sont donc pas intimement soudés les uns aux autres mais seulement mis en contact. Aussi est-il possible qu'une partie de l'efficacité de ce système ne puisse être immédiatement contrôlable. Cette réserve a déjà été formulée notamment pour expliquer l'incohérence de certains résultats des expériences 1, 11 et 6.

Sur l'homme la cicatrisation est complète lorsque les résultats audiométriques sont enregistrés. Cependant il existe souvent bien d'autres facteurs que la valeur de la masse ossiculaire restant influençant la réponse. Citons en particulier la fonction nasale et tubaire, les adhérences cicatricielles anormales dans la caisse ou l'attique gênant le système tympano-ossiculaire créé.

Malgré ces réserves, de telles expériences nous paraissent utiles au chirurgien. Il semble indispensable qu'entre autres facteurs, celui-ci cherche avant tout à résoudre au mieux le problème physiologique pur et obtenir par son acte opératoire, une oreille moyenne dont les caractéristiques soient le plus proche possible de celles d'une oreille normale.

Cette règle générale s'applique en particulier au respect de la masse ossiculaire dont la suppression même dans de bonnes conditions tympanoplastiques perturbe considérablement la phase de l'onde arrivant à la cochlée.

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45 Cours du Mar chal Foch Bordeaux

ECHO LOCATION IN BATS

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The skulls, temporal bones and cochleas of some insect-eating night-feeding bats have been examined. The minute structure of the cochlea is considered with regard to the use of ultrasonic waves and their echoes for locating obstacles and prey in complete darkness. A theory as to how these animals find their way through obstacles to their food is put forward.

In the year 1793 Spallanzani in Italy discovered that bats, while in complete darkness, were able to find their way about, were able to catch their prey—in the form of small flies and moths—on the wing and were able to avoid obstacles. He found that blinded bats were not handicapped and fed as well as their seeing companions. He found, however, that if the ears were blocked, the bats were liable to collide with each other and with obstacles and were unable to catch their food. Such bats were quite helpless in the dark and it was obvious that they depended on their hearing for navigation and for hunting. Although these findings were confirmed by others, the suggestion that bats used their ears to find their way about was not accepted by the scientists of the time, but no other explanation was put forward until Hartbridge in 1920 suggested that bats used the same method of locating objects as had been developed for detecting submarines during the first world war—by sending out signals and noting the time of arrival of the echo. Hartbridge suggested that the signals sent out were of a frequency above the range of the human ear but it was not until 1938 that Pierce & Griffin by means of electronic measuring equipment proved that the calls made by bats were of frequencies well above the possibility of human hearing. They do, in fact, employ sounds of from twenty to one hundred or more kilocycles per second.

It must be recalled at this point that bats or Chiroptera are divided into Megachiroptera or the fruit-eating bats and Microchiroptera which feed exclusively on insects. The former, which do not fly or feed in complete darkness and whose food is stationary, do not use the acoustic method of navigation.

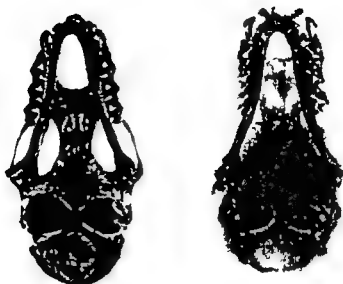
The Microchiroptera bats are smaller and are found in their various species throughout the world. They have large external ears, some of them very large and in some species the ears are capable of movement in many directions. The inner ears in these bats are specially developed for the very high frequency of the calls and the low intensity of the echoes and the cochlea is

It is to be reported in this document has been sponsored in part by the Air Force Office of Scientific Research of the Air Research and Development Command, United States Air Force through its Liaison Office.

Vespertilionid larynx $\times 7$ Rhinolophid larynx $\times 7$

FIG. 1

proportionately larger than in the average mammal. In some, especially the *Rhinolophus* or horse shoe bat the two cochleae are so large that they are almost in contact in the midline and occupy a large proportion of the posterior compartment of the skull (Fig. 2). The basilar membrane is very short and the spiral ligament is extremely well developed and continues in the part bordering the scula tympani an additional bony lamina—the external spiral lamina (Fig. 3). This cochlea is obviously admirably adapted for the reception of sounds of very high frequency. The sounds which are emitted by the animal itself are formed in the larynx and projected either through the mouth or the nostrils. It had been suggested that these sounds were not formed in the larynx but by some other part of the body but it was found that covering the mouth—or nose—had the same effect as blocking the ears. Some surprise had been expressed that sounds of such very high frequency could come from a mammalian larynx. The first description of the interior of the larynx by Robin (1891) included two folds on either side wall of the larynx. Ellis (1907) described superior and inferior vocal cords and made drawings of these structures. Recent examination of the larynx of bats has shown that the larynx is unusually large and has a series of very powerful extrinsic muscles on its lateral aspects. The epiglottis and glottis are somewhat small for the size of the larynx but follow the usual pattern of the mammalian larynx. The interior of the larynx, which is relatively more capacious than that of the human species, shows on either side a vocal cord, a ventricle and a *ventricular band* as in any mammalian larynx. The vocal cord is relatively short and massive and its long axis is directed upwards and forwards from the arytenoid region (Fig. 1).

Fig. 2. *Myotis* skull $\times 20$.

It has been accepted for many years that bats used echo location during their activities in the dark, but it has not been understood as to how this was actually achieved. Various theories have been put forward but all have suffered from objections which have invalidated them. An explanation, which appears at least plausible, of the methods by means of which two different types of bat make use of the echo from their calling signals to catch their food whilst both are in flight and to avoid all obstacles in complete darkness is put forward. Among the types of bat which are common in Europe and in North America there are two main groups which employ entirely different methods of locating prey and obstacles.

1. *Vesperugo* The members of this group have external ears which face forward and do not engage in movement independent of the head. These bats emit a signal or call of 30-70 kilocycles per second, which is of short duration, 1-4 milliseconds. The frequency falls during this period by an amount which may be as much as an octave but rarely more.

2. *Rhinolophus* The so-called horse shoe bats because of the leaf-like structure in the shape of a horse shoe on the nose. These bats have external ears which are capable of considerable independent movement relative to the head and to each other. They emit a signal or call which is of much longer duration, 40-100 milliseconds and of which the frequency is very high, 80-100 kilocycles, and remains constant throughout the pulse except for a fall during the final two milliseconds of not more than a half octave.

If a call were sent out with uniform frequency and reaching an object returned as an echo, the echo would overlap the call and would probably not be recognisable. It would in any case be of similar frequency to the call and would give little information as to the nature or position of the object.

FIG. 3 *Rhinolophus* cochlea $\times 110$

Since the energy of the outgoing pulse is very high it may be supposed that the bat's ear is overloaded and received signals are distorted. If this is so a difference in frequency between the call and the echo will give rise to beat notes audible to the bat. These beat notes may be shown to give accurate information about the position of the object returning the echo. Both groups of bats use the call and echo but each varies it in a different way so as to gain the required information.

The *Vespertilionid* sends out a call which falls in frequency as much as an octave in a period of not more than four milliseconds. As bats operate the echo location at short distances the echo returns while the call is still being emitted and there is thus an overlap. The overlap is not however complete as the echo may be two or more milliseconds later than the call and so the highest frequency of the echo overlaps the call when its frequency has already decreased. This gives rise to a beat which is readily detected by the bat and by the human ear if distortion is artificially introduced. The greater the delay in the return of the echo the higher will be the frequency of the beat and as the bat approaches the object the frequency of the beat at any given instant will indicate to the bat the distance of the object (Fig. 4). The beat finally disappears as the bat reaches the object.

The same mechanism may also give directional information. If an object lies to one side of the bat the time of arrival of the echoes in the two ears will be different. A comparison of the beats heard in each ear will then decide the angular direction in which the object lies. The mechanism suggested would be therefore binaural and it has been demonstrated that a *Vespertilionid* bat with one ear plugged is completely helpless.

The *Rhinolophus* bat depends on the Doppler effect on the echo of his call which is caused by the relative velocity with which he and the object are

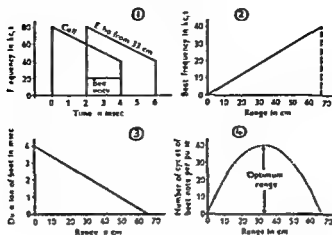


FIG. 1

approaching each other. The Rhinolophid emits a call of uniform frequency but of longer duration and as he approaches the object the frequency of the echo will be raised (Fig. 5) and beats will be heard and the frequency level of the beats will indicate the relative speed at which the object is being approached. In these bats an estimate of direction and possibly of range is provided by voluntary movement of the ears. In addition to these there are ear movements at a rate of 50 cycles per second which are sufficient to produce secondary Doppler effects on the echoes from stationary objects.

A groove in the leaf-like structure on the nose which gives this family its name may serve to carry sound from the nostrils to the immobile base of the ear. This sound is of unvaried frequency and when compared with the variations in the frequency of the echoes which are caused by the rapid movements of the pinnae will produce further beats and help considerably in location. These mechanisms do not require the use of both ears and it is found that a Rhinolophid with one ear plugged flies as expertly as the normal animal.

When a bat is searching its locality for food or an obstacle it sends out calls at relatively infrequent intervals but as soon as an object appears on the acoustic horizon the rate at which the calls are emitted is stepped up and the echo location operation is focussed on the recognised object.

The degree of discrimination of which these methods are capable is such that the presence and position of several objects may be estimated at the same time. As an instance bats are frequently observed flying above the surface of water or of the earth—without striking either, pursuing and catching an insect and avoiding all obstacles at the same time.

Hitherto it has not been clear how faint echoes may be heard by the bat while he is making his very loud calls. The theory put forward attempts to overcome this difficulty since it demands overloading of the ear and does not require that the echoes be heard as such. A bat listening to beat notes could obtain much information simply by noting their pitch.

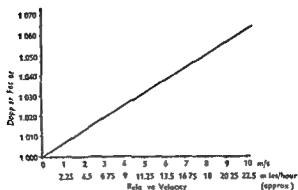


FIG. 5

It has been noticed that a slight clicking noise can be heard by the human ear when in the proximity of a bat. This is caused by a very short pulse of low frequency sound emitted by the bat immediately before sending out his call perhaps some sort of warming up action.

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DISCUSSION

G. Kelemen. Démonstration des coupes du larynx du chauve-souris tropical *Hypsignathus monstrosus*.

OTOSCLEROSIS IN OSTEOGENESIS IMPERFECTA

A Study in the Etiology of Otosclerosis

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For more than a decade otologists have been occupied with devising ingenious and complicated operations to relieve deafness caused by otosclerosis. They have thus lost sight of the principle that the scientific method of investigation of disease is to endeavour to discover the cause and thereafter to devise a cure based on precise knowledge of etiology and pathology. This communication is presented in the hope that it may stimulate interest in those aspects of the disease which will assist in the discovery of its origin.

Theories of Causation

There have been many theories regarding the etiology of otosclerosis its pathological development and its mode of spread. Broadly these theories can be classified in three main groups.

1. Those which regard otosclerosis as a form of embryological disturbance having its origin in some embryological abnormality.

2. Those theories which depend upon a connection with some nervous or sympathetic imbalance.

3. Those theories which regard the disease as inflammatory and based upon a circulatory disturbance.

Sichenmann, one of the earliest students of the pathology of otosclerosis regarded the condition as a renewed activity and a continuation of the normally interrupted phase of cartilage in the ossification of the labyrinth capsule. He considered it therefore a continuation of growth beyond normal limits.

More recently Luggenheim stated a regression theory believing that the parts affected are those most recently acquired and that the site of predilection namely the area of the fossula is a homologue of various accessory ducts in the lower vertebrae.

Otto Mayer has suggested that excessive strains play a part and that there is a resorptive failure of primitive reticular bone.

Gray has several theories but one of his most interesting is that the essential factor is a slowly progressive failure in the function of the vasomotor reflex of the organ of hearing as a whole. He stated his belief (1934) that there is degeneration of the nerve fibres starting with the medullary sheath.

Ozay in this connection demonstrated that the functional loss of the cervical sympathetic in guinea pigs leads to retrogressive changes of the auditory organ.

Gottlieb also considered that the pathological changes in the capsule and

changes in the auditory nerve are produced through neurovascular disturbance

From the point of view of the inflammatory theory, Minasse Wittmaack & Fraser believed that inflammation for one reason or another was the essential cause. Weber supported this view and thought that there might also be another constitutional factor probably metabolic, which might be an hereditary or transmissible tendency. More recently Wolff has followed up the theory of Wittmaack that congestion and stasis are the primary causes of bone change, and has advanced histological evidence which she believes supports this theory.

Lastly Nager & Mayer believed that there is a close connection with other bone diseases, indeed Nager has called otosclerosis a localised Paget's disease. They admitted that this was a difficult theory to sustain owing to the lack of evidence of disease elsewhere in the skeleton. Bunch (1934) has pointed out however that there has been little systematic effort to verify this.

Early Stages

Many workers realised the necessity of attempting to find the earliest stages of otosclerosis. Wittmaack attempted to work backwards from the established forms of otosclerosis to what he believed to be the earlier stages.

Stacey Guild's contribution (1930) was a valuable description of changes seen in the bone of eight cases at an early age but as there have been claims to identification of otosclerosis in the foetus these do not necessarily represent the earliest forms of the disease.

The blue mantle has been accepted as an essential part of otosclerosis, and Empert & Wolff (1949) suggest that they are merely clinical indicators of the type of bone being laid down.

Weber (1933) however supported the idea that blue mantles are otosclerosis in its early stages but I. P. Fowler Jr (1934) pointed out that blue mantles were encountered in *otitis media*. In his studies of the ossicles Cowell also found mantling but not otosclerosis in its accepted form.

In the Edinburgh collection blue mantles have been seen in the earliest stages in manifest otosclerosis and in very long standing quiescent disease.

In the study of otosclerosis the fenestration operation afforded an opportunity to obtain unlimited material. There was the added advantage that the clinical history and the details of examination were known and could be correlated if necessary with the histological findings.

The first stage in the investigation was the collection of material and its classification and the results of this investigation have been published elsewhere (Ogilvie & Hall 1953). This gave a groundwork and the development of the disease assumed a definite pattern.

In all stages evidence of healing bone was observed and in no case was there evidence of breakdown of bone with the exception that in one or two sections a small degree of osteoclastic activity was seen. This appeared to be

a concluding process and not an active one as the osteoclasts were few and degenerated. This finding confirmed the impression that at some stage osteoclastic activity and the breaking down of bone must be the predominant feature of the disease.

One of the most important points is that with the exception of the opinion of Gray osteoclastic activity must play a part: no support could be found in the Edinburgh researches for any of the theories which have been mentioned. No evidence could be found of any renewed activity.

There was no progress of the disease along blue mantles and no advance on a broad front by any known and accepted pathological process.

Indeed it was possible to prove that the blue mantle was part of the healing process and can be shown to have all the characteristics of healing bone. Examination of sections showing a line of demarcation failed to show any evidence of advancing disease by osteoclasts such as would have been expected.

Correlating the clinical findings with the histological appearances it became evident that healing of bone indicated increasing deafness. Otosclerosis therefore appeared to be a process of healing bone and not a disease entity.

The problem thus resolved itself into a question of the cause of the healing and the discovery of the condition which made such a process necessary.

Hereditary influences were evident in otosclerosis so attention was directed towards the bone dystrophies and in particular to Nagel's suggestion that otosclerosis is a localised Paget's disease. This idea was soon discarded. In the comparison of any diseases it is necessary to consider the whole disease and not to draw conclusions from comparisons of histological preparations and although there was some similarity between the diseases histologically the age, sex and distribution of the diseases showed that they were dissimilar. Our attention was then directed towards the points of resemblance between osteogenesis imperfecta and otosclerosis. That 10-60% of all cases of osteogenesis imperfecta show otosclerosis is a known and accepted fact.

That this is a true otosclerosis was shown clearly in conjunction with Professor Williamson in a study of these diseases in a mother and daughters (1933).

Having confirmed the facts of the identity of the diseases and their transmissibility there were still some very difficult problems to solve such as the mode of spread, the irregular location of the disease, the time of onset, the activation of the disease and its varying degrees of severity.

These were difficulties which for a time seemed insurmountable till it was realised that if sufficient early cases of osteogenesis imperfecta could be discovered and examined then it was probable that a case would be found which would have the earliest stages of otosclerosis and in this way a solution to the problem might be found.

Within a short period the necessary material was obtained and some interesting facts were discovered.

It immediately became apparent that even at birth or before it there is activity in the bone of osteogenesis imperfecta. The imperfect development

of the osteoblasts induces an instability of the bone which immediately undergoes osteoclastic absorption and replacement with healthy bone. This occurs in several localities at the same time and varies greatly in degree and extent, at times appearing in small isolated areas whilst at others the process is widespread.

Moreover it was evident from the material available that the area surrounding the oval window was particularly vulnerable not merely to changes within the bone itself, but also to influences from the middle ear. There was therefore a rational explanation of some of the problems. A stage of widespread osteoclastic activity had been discovered. The explanation for irregular and isolated foci of disease was evident and the vulnerability of the region of the oval window was demonstrated.

In many cases areas of healing bone would heal completely and there was no reason for suspecting their presence; in others the disease might be more widespread and in particular might involve the oval window and so produce fixation of the stapes and therefore would call attention to itself by causing deafness.

Osteogenesis imperfecta is known to provide variations in degree of severity and distribution and it would appear that severity of symptoms can vary within very wide limits.

There are without doubt several important details to be filled in. One of the most important of these is the question of the influences which cause such wide differences in the severity of the changes in otosclerosis. It is suggested that several factors may be responsible.

These may be inflammatory, for it has been shown that the area surrounding the oval window is particularly exposed to the effects of inflammation in the middle ear. Endocrine disturbances may well play a part and psychosomatic influences cannot be excluded.

Further evidence is required to determine which or how many of these conditions play a part and to what extent.

It would seem therefore that otosclerosis is fundamentally an osteoblastic insufficiency and has the same basic etiology as osteogenesis imperfecta differing only in degree, extent and location.

In the most severe forms it causes collapse of the skeleton and death. It varies through all grades of severity to the stage where attention is called to it only because it occurs fortuitously in a situation where, by interfering with the middle ear mechanism, it causes deafness.

The coloured slides and photomicrographs with which this lecture was illustrated were prepared by T. C. Dodds I.R.P.S., of the Photographic Section of the Pathology Department of the University of Edinburgh.

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DISCUSSION

A. Seeger: Zu dem sehr interessanten Vortrage vom Herrn S. Hall moechte ich mir eine Frage erlauben. Er moege mir entschuldigen wenn ich ihn schlecht verstanden

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THE EFFECT OF LOCAL IRRADIATION OF THE LABYRINTH IN THE RAT WITH IONIZING PARTICLES

A Preliminary Note

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During the last decade ionizing radiation has been used to produce restricted lesions in animal tissues. This method has been applied to destroy the hypophysis (cf. Notter 1959) or nervous tissue (cf. Larsson *et al.* 1958). Prompted by earlier attempts to produce local labyrinthine effects by diathermic cross fire irradiation (Lisholm & Nilén 1925) Nilén has (1959) suggested the use of ionizing radiation in oto-microsurgery as a complement to mechanical operations. This idea will be discussed in brief on the basis of the results of some attempts to bring about local destruction of labyrinthine tissue in the rat by means of a narrow beam of high energy protons. We spoke about this at the 14th Scandinavian Otolaryngological Congress in Stockholm in June 1960 (Nilén *et al.* 1960).

The irradiation was performed with a 2 mm wide 185 MeV proton beam from the synchrocyclotron at the Gustaf Werner Institute at Uppsala. This beam is useful for quantitative studies of the effects of local irradiation because of low scattering and a favourable depth dose curve (cf. Larsson *et al.* 1959). It is therefore possible to irradiate small volumes of deep lying tissue uniformly. In these experiments the beam was directed so as to destroy parts of the right labyrinth passing in the dorso-ventral direction through the skull of the immobilized animal (Fig. 1). In other details the radiation technique was as described by Larsson (1960). The absorbed radiation dose was 10 krad.

Twenty rats were irradiated. They were killed at different times up to six months after irradiation. Depending on the site of the lesion either no or the following symptoms were noted: e.g. head deviation and manège gain to the right, decrease of tonus on the right side, nystagmus to the left or deafness in the right ear (Fig. 2).

So far only three of the animals have been investigated histopathologically. In one of these, killed two weeks after irradiation, some few red blood corpuscles were found and oedema was suspected around the stapes, indicating early changes. In the second rat, killed eight weeks after treatment, the beam had induced degenerative damage of the right acoustic nerve just inside the internal acoustic foramen and no pathological changes were noted in the inner



Fig. 1



Fig. 2

Fig. 1. Autoradiogram of the proton beam—the light circular spot—superimposed on a roentgenogram of a ratskull in the region of the right labyrinth (negative picture)

Fig. 2. Typical x-ray is of acute destruction of the labyrinthine functions on the right side after irradiation. The rat was killed eight weeks after treatment.

ear or other parts of the temporal bone. In the third rat killed twelve weeks after irradiation there was well circumscribed damage of bone and muscles just outside the middle ear. Part of the damaged bone was completely necrotic (Fig. 3).

From this small series the following conclusion may be drawn:

A proton dose of 30 krad applied in the way described is able to induce sharply defined damage of bone or peripheral nerve tissue within two months after irradiation with only slight reaction in the surrounding tissue. Our experiments do not, however, permit definite conclusions to be drawn concerning the reaction of the labyrinthine capsule to proton radiation, as this tissue has a structure that is somewhat different from bone tissue elsewhere. We believe, however, that the changes will be about the same as in Fig. 3.

It is of course too early to answer the question whether ionizing radiation can be used for clinical otomicrosurgery. Our observations might, however, stimulate further work in this field. Supplementing the mechanical knife by locally applied necrotizing radiation would certainly be of great advantage as this would increase the chances of destroying or inhibiting the primary focus of the otosclerotic process. Certain other diseases of the inner ear and its central connections might also be tackled by this means. Our experiments are continuing on a broader basis.

As to the question of the most suitable radiation source for practical use in the clinic the best choice would probably be a preparation of a suitable



FIG 3 This picture from a rat killed 12 weeks after irradiation and without any clinical symptoms shows the well limited total dissolution of the bone and muscles outside the middle ear*. The bone around the defect is irregular and strongly basophilic coloured. More peripherally the marrow lacunae are filled with fibrous tissue rich in cells.

radioactive nuclide such as Yttrium 90. Applicators of this convenient beta emitter which can deliver doses of the correct order of magnitude within an hour or less can be produced today. Such an applicator could be put close to the target via the external meatus after tympanotomy or via the Eustachian tube.

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DISCUSSION

E P Fowler, Jr Similarly we propose to Prof Nylen that the changes produced by radiation are initiated by vascular changes and that the neural changes are secondary to these. Even with lethal body radiation there are hemorrhages into the labyrinth such as he mentioned with local application. I doubt therefore that radiation is a reasonable treatment to try on otosclerosis—If anything, radiation would simply produce a bone necrosis but, with healing it might well in the end make the condition worse.

I Simpson Hall (to Fowler) Dr Fowler's criticism that some special mechanism of blood vessels or some psychosomatic disturbance did not affect the present ideas in fact, they were complementary to them. Dr Fowler knew just as well as Dr Hall that severe emotional stress, vasomotor influences, and other causes, including great hormonal changes, could influence the progress of almost any process in the body and he saw no contradiction in the fact that the reparative processes were stimulated by such disturbances.

C O Nylen (Reply) I can well imagine the theory of Fowler that the small vessels are engaged and damaged at first by irradiation with high energy particles. The works of Larsson *et al*, where nervous tissues has been treated, show it. However, all the rats without symptoms lived a normal life after irradiation, before they were killed.

THE IMPORTANCE OF HEAD MOVEMENTS IN STUDIES INVOLVING STIMULATION OF THE ORGAN OF BALANCE

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Our interest in this field of research started a few years ago from investigations into the basic physiology of motion sickness. A search of the literature and preliminary investigation indicated the significance of the non-auditory membranous labyrinth in this condition.

Evidence for this close relationship is direct and mainly based on these facts:

(a) Animals which are susceptible to motion sickness are immunized by removal of the vestibular organs or by dividing the eighth cranial nerves. This was first done in dogs and subsequently verified in cats (McVally & Stuart 1942).

(b) Ablation of the vestibular tracts in the cerebellum eliminates motion sickness in dogs (Tyler & Bard 1949).

(c) Deaf mutes with a congenital absence of the non-auditory labyrinth are known to be immune (Tyler & Bard 1949).

(d) Patients whose labyrinths had been destroyed by disease are immune (Graybiel 1936).

Furthermore, it is well known that patients whose labyrinthine sensitivity is being determined by caloric stimulation of sufficient strength to cause marked nystagmus, often also exhibit the pallor, cold perspiration and nausea characteristic of motion sickness.

The importance of measuring head movements as a means of determining the characteristics of vestibular stimulation has been brought to our attention forcibly in our research dealing with the basic physiology of motion sickness and spatial orientation (Johnson 1946; Johnson *et al.* 1951) when it was established that, unless the head is firmly fixed to the vehicle, the accelerations to which the end organs are exposed are never the same as the motions to which the trunk is exposed. Failure to realize this can readily result in erroneous conclusions. Thus, for example, Manning was unable to cause motion sickness in an elevator when the heads of his test subjects were fixed to the seat, while Wendt & McEachern were able to make their subjects sick by similar vertical accelerations of the trunk but without head fixation.

It is the object of this paper to describe the procedures and findings which signify the requirement for head movement measurements as a means of determining the resultant accelerations which constitute the actual stimulation



FIG. 1. Miniature gyroscopes attached to helmet in each of the three orthogonal planes enable precision recording of head movements.

PROCEDURES

Recordings of head movements were first made mechanically on a two pole swing which exposed the subjects (head free to move and subjects blind folded) to simple harmonic motion. A linen thread passing through a pulley was connected from the subject's head to a pen which recorded the movements on graph paper rotating on a kymograph. The recording was roughly calibrated by deliberately tilting the head forward through 10 degrees (the atlanto occipital joint was taken as the fulcrum for these movements) and the upward displacement of the recording pen was impressed on the graph paper in arbitrary units, each of which represent one tenth of an inch and is equivalent approximately to one degree of angular deflection when occurring in the plane of the swing (1948).

In order to obtain more accurate measurements both qualitatively and quantitatively a robot organ of balance was devised. By the attachment of miniature gyroscopes to the head of our subjects (Fig. 1) it was possible to record the resulting angular movements of the head in three different orthogonal planes (relative to the body and to the ground). This apparatus has been used both in the laboratory and during flight to record the resulting angular accelerations in each of the three orthogonal planes of space. More recently linear accelerometers have been added.

In order to control the magnitude and direction of forced angular acceleration acting on the non auditory labyrinth it was first decided to place the body horizontally in the supine position in order to facilitate simultaneous rotation of the trunk together with forced head movements in one of the other orthogonal planes of space. As a first attempt a stretcher was mounted on a

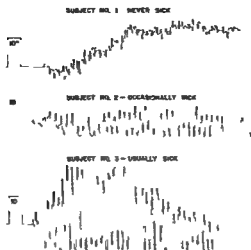


FIG. 2. Recordings of head movements independent of the swing of three different subjects swung through a 70-degree arc with a frequency of 16 cycles per minute. The swing was hand propelled.

motor driven turntable and the subject was instructed to turn his head from side to side while the body was being rotated about a vertical axis. The rate of rotation of the stretcher was 30 rpm which is an established rate well above the threshold of the average individual for stimulation of the semicircular canals by sudden de acceleration. We now place the subjects in the sitting position and superimpose controlled nodding movements of the head while rotation of the trunk occurs around a vertical axis.

Another procedure which we have recently adopted consists of revolution without rotation. This involves the use of an eccentrically mounted counter rotating secondary turntable. A full description of this device is to be published shortly. Essentially the subject travels in a circular path (revolves) in a horizontal plane around a central point but remains constantly facing in the same direction. In this situation the head and trunk are exposed to linear accelerations only, thereby stimulating the otoliths and not the semicircular canals.

RESULTS

A. Two pole Swing Experiments

The recording of head movements of human subjects during runs on the hand propelled swing showed that there are variations in the pattern of head motion on the body chiefly in its sagittal plane. This is indicated in Fig. 2, which shows a recording of the head movements of the three subjects.

It is most interesting to note that although the three individuals were subjected to similar conditions of swinging the one who showed the least independent head movement had a history of being very resistant to motion sickness,

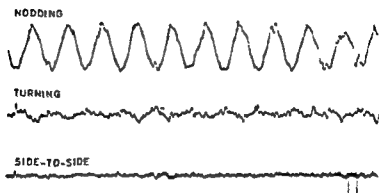


FIG. 3. Gyroscopic recording of the angular accelerations of the head in each of the three orthogonal planes of a subject seated on swing.

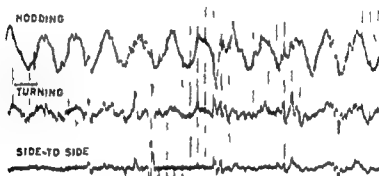


FIG. 4. Gyroscopic recording of the angular accelerations of the head in each of the three orthogonal planes of another subject seated on swing.

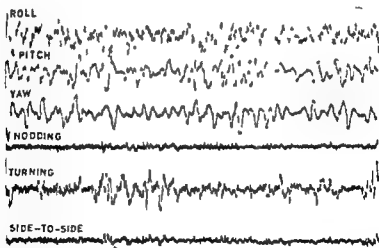


FIG. 5. Simultaneous gyroscopic recording of the angular accelerations of a C-45 aircraft (roll, pitch and yaw) and of the head of the co-pilot (nodding, turning and side to side) during moderately rough turbulence.

C. Revolution without Rotation

At the time of writing, our experience with the counter rotating secondary turntable is much less than with the other devices. Evidence to date, however, indicates that it is ineffective in producing motion sickness, even if the subject's head be allowed to move in one plane. No vertiginous sensations or records of nystagmus have been produced. The eye movements which do occur indicate otolith stimulation. Further research is planned with this procedure and the results will be reported at another time.

SUMMARY AND CONCLUSIONS

Certain findings and procedures have been described which enable precise determinations of the sensitivity of the various components of the non auditory membranous labyrinth.

We wish to emphasize very strongly one point which applies to motion sickness induced by various devices. The angular and linear accelerations that have been described as characteristic of various pieces of apparatus refer to the apparatus alone. If valid conclusions on the effects of these accelerations are to be drawn, care must be taken to ensure that the motion of the subject's head conforms as closely as possible to that of the apparatus. If the head is allowed to move freely, either voluntarily or involuntarily, it will be subject to additional accelerations due to these motions, and, in particular to angular accelerations because of the short radii involved in head movement with respect to the body (rotation of the head in the horizontal plane or swinging anteroposteriorly or laterally on the neck).

It is hoped that the procedure outlined will be of use to those interested in vestibular threshold determinations both from the point of view of basic physiology and in the diagnosis of vestibular disease.

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DISCUSSION

1. Meyer zum Gottesberge. Vor langen Jahren untersuchte ich die Wirkung der Zentrifugalkraft auf den Vestibularis in einem „Rotor“, einer mit grosser Geschwindigkeit sich drehenden Trommel wie man sie auf Jahrmärkten findet. Solange der

Kopf nicht bewegt sich, sind die vestibulären Reaktionen erträglich. Bei einer Kopfbewegung während der Drehung tritt sofort heftiger Schwindel und Nausea auf.

H. Frenel: Der Film von Herrn Johnson stellt eine ausgezeichnete Illustration der Coriolisbeschleunigungen dar, die während einer Drehung dann auftreten, wenn Massenteile der Drehachse genähert oder von ihr entfernt werden. Wenn ich mich recht erinnere, so hat Wojatschek vor vielen Jahren eine Prüfung auf Verträglichkeit der Labyrinthreize bei der Fliegenergungsprüfung angegeben, die sich der Coriolisbeschleunigungen durch zusätzliche Kopfbewegungen während einer Drehprüfung bedient.

R. Müllermeier: Die Weltraumschiffe, die die Erde später umkreisen werden sollen, sich soweit ich weiss wegen der dort herrschenden Schwerelosigkeit in ständiger kreiselnder Bewegung befinden. Kann man schon etwas darüber aussagen, wie rasch diese Drehbewegung sein soll oder sein darf, damit die dort lebenden Menschen nicht durch Coriolisbeschleunigungen in ihrem Gleichgewicht gestört werden?

I. P. Coulter, Jr.: As for Dr. Johnson's beautiful but elaborate apparatus for stimulating the macular organs without stimulation of the canals, may I suggest that excellent stimulation can be obtained in any plane by rotation for a long enough period with subthreshold angular acceleration (less than 0.5° per sec)?

I. B. W. Jongkees: The contribution of Dr. Johnson seems to be very important for both aviation medicine and vestibular physiology. Nevertheless some questions remain open.

Is the use of a glass model of a much bigger size than that of the real labyrinth really enough to prove the scheme of movements in our labyrinth with its capillary canals?

Why does Dr. Johnson use such a complicated system to stimulate the otoliths? A rotation at constant speed is a perfect stimulation of these organs by way of the centrifugal force.

Finally I have to make an objection against one sentence used by the speaker. The expression "statistically significant and therefore not due to chance" is untrue.

It is not probable. It may even be extremely improbable that the effect is due to chance but not impossible.

U. Surala: To Dr. Johnson I would like to add that in this connection we have not counted the eosinophilic cells.

W. Johnson (Reply): Mister Chairman, I wish to thank all those who discussed my presentation. It is a healthy sign to hear constructive criticism expressed. Allow me Professor Arslan to express my gratitude for the invitation of the Collegium which enabled me to be present and to meet so many of the world's outstanding names in Otolaryngology.

ÜBER DIE STATOAKUSTISCHEN UND PSYCHOSOMATISCHEN STÖRUNGEN DES FLUGHAFENPERSONALS DURCH LÄRM

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Der schädliche Einfluss des Lärmes durch tiefe, mittlere und hohe Töne durch Propeller Flugzeuge verursacht sowohl statoacustische als auch psychosomatische Störungen.

Die Jet Flugzeuge verursachen ebenfalls solche Störungen, welche aber viel frequenter und stärker beobachtet werden. Diese werden einer ultrasonorischen Einwirkung zugeschrieben.

Im Rahmen eines kurzen Referates mochten wir mit einer Anzahl von Bildern dieses Thema behandeln.

Wie bekannt erzeugen die durch kolben Propeller angetriebenen Luftschiffe einen starken Lärm der 116-125 db und eine Frequenz von zirka 100 Hz. Dies hängt von der Maschine als solcher ab, ob die betreffende sich im Leerlauf oder in vollem Betrieb während der Messung befindet. Die Ausbreitung des Lärmes hängt von verschiedenen Faktoren ab, die mit der Umgebung verbunden sind, z. B. Hangar, die Nähe von Gebäuden etc.

Dieser Lärm ist zwar stark und störend, aber nicht mehr als der Lärm in der Schwerindustrie mit seinen bekannten Konsequenzen bezüglich des Hörapparates.

Anders stehen die Dinge und die Verhältnisse samt ihren Auswirkungen bei den Bewohnern der Nähe eines Flugplatzes, die im Schlaf gestört werden bezüglich ihres psychosomatischen Zustandes.

Eine zweite Art von Luftfahrzeugen stellt die Kombinationsform von Propeller und Düsenmaschine dar. Diese erzeugen einen akustisch schwachen Lärm, aber produzieren durch die Düsen Ultraschallschwingungen. Diese Motoren erzeugen bei vollem Betrieb einen Lärm von 140 db und eine Frequenz von 200 Hz Ultraschallschwingungen. Letztere werden von Mikrophonen des Ichometers nicht aufgefangen, so dass sie den Apparat überhaupt nicht erreichen. Deswegen bedienen wir uns eines geeigneten Kontakts und Kristallmikrophons, um die Ultraschallschwingungen auf den Oszillographen zu übertragen und optisch darzustellen. Aber dies war nicht befriedigend.

Eine dritte Art von Luftschiffen betrifft zwei Kategorien, die Düsenflugzeuge vom Typ Caravelle, die sehr starken Lärm erzeugen (140 db) und diejenigen, die durch Abschirmung den Lärm dämpfen, z. B. Comet. Bei den ersten ist man schon instinktiv gezwungen, sich vor dem Lärm zu schützen. Beim zweiten Typ wird der Lärm ca. um die Hälfte herabgesetzt, wirkt da-

durch nicht unerträglich und man ist ihm somit stärker ausgesetzt. Da die Ultraschallschwingungen aber noch in starkem Masse vorhanden sind, wirken sie die oben erwähnten psychosomatischen Störungen, die höchst wahrscheinlich als Spätschäden konstatiert werden und zwar betreffen sie die innersekretorischen Drüsen, das autonome Nervensystem und haben Auswirkungen auf Herz-Kreislauf usw. wie diese experimentell, statistisch und klinisch schon von verschiedenen Autoren beschrieben wurden. Deswegen glauben wir, dass in den nächsten Jahren durch den vermehrten Düsenflug Betrieb trotz der Abschirmung oder besser gesagt gerade wegen der Abschirmung und der Verminderung des akustischen Lärmreizes nicht auszuführen vorsichtsmässig ihnen gegen die U.S.S. werden wir die Opfer der Ultraschallwirkungen in grosserer Zahl zu sehen bekommen.

Solange es noch Zeit ist, soll man durch gesetzliche Massnahmen die Bevölkerung schonen und Richtlinien zur Entschädigung der Opfer, die für ärztliches Gutachten festlegen, die zum Kreise des Boden- und Buropersonals eines Flugplatzes zählen.

Der Lärm hängt noch von der Fliegerart z.B. ob das Flugzeug steil aufsteigt oder mehr horizontal, ob die Maschine im Vollauf gefahren wird, von der Pferdekraft der Maschine, ob das Flugzeug vollgeladen ist und schliesslich von der Windstromgeschwindigkeit und von noch anderen Details, so z.B. je nachdem ob sich die Messperson unterhalb, vor, hinter oder seitlich vom fliegenden Aeroplan befindet.

Bekanntlich kann die akustische Überlastung, Hörermüdungssymptome hervorrufen. Ebenfalls kann die akustische Überreizung einen Übergang von der physiologischen zur pathologischen Ermüdung des Ohres führen mit oder ohne definitiven Funktionsverlust, der auch reversibel sein kann. Das akustische Hörtrauma zeigt eine Stimulationsabhängigkeit mit individuell veränderter Hörschwelle und ist in einer Pathologie der Haarzellen oder in einem Einrissen der Membranen (Reissner-Basilaris) begründet. Die Ursache kann durch einen Ton — wie bei unseren Versuchen —, als auch durch Lärm bzw. Erschütterungen z.B. bei Fliegern hervorgerufen werden. Nach *Rudi Daxner and Marie Jung* hängt dies von der Intensität und der Dauer ab. *Unutzer* Meinung nach ist die Schwingungszahl (Hz) besonders bei hohen Tönen ebenfalls ausschlaggebend (Ultraschall). Das akustische Trauma etabliert sich meist gegen 65 und innerhalb von 4000–8000 Hz. Dort findet man Hörlucken oder bei ausgedehnten Schädigungen nur Hörinseln. Von Gool und Van Dethoch haben sich der kontinuierlichen Audiometrie durch das Audiometer von Bekesy bedient, wir haben das japanische Audiometer benutzt. Die kritische Intensität liegt nach *Rudi und Hoop* bei 95 db von 2048 Hz. *Isser* betont, dass eine individuell verschiedene Hörschwelle überschritten werden muss, um einen Reiz bei einem reversiblen oder irreversiblen Hörtrauma bei einer Einwirkungsdauer von einigen Minuten zu bewirken. Dabei kno-

1. *AN 8* a) frequency oscillator, Kasuga Musen Kogyo Co. Ltd. Wlnhor model 3) A cathode ray oscillograg. time base.

ligt die Erholungsdauer bei solchen experimentalen Belastungen ca 48 Stunden

Unserer Meinung nach besteht ein Zusammenhang zwischen dem vorderen und dem hinteren Labyrinth der sich besonders bei ganz hohen Tönen von über 20000 Hz bemerkbar macht die zwar vom vorderen Labyrinth nicht wahrgenommen werden jedoch dasselbe angreifen und für das hintere Labyrinth ausschlaggebend sind. Es besteht somit zwischen dem vorderen und dem hinteren Labyrinth bei einer adäquaten Reizung des vorderen Labyrinthes eine Abhängigkeit die wahrscheinlich durch die direkte Nervenfaserverbindungen oder durch Überspringen des Reizes oder durch zentrale und allgemeine Reizung bedingt wird.

Nach einer Belastung schon von 20 db hatten wir nach einer Viertelstunde Ermüdungserscheinungen die ca 48 Stunden dauerten. Bei Arbeiten im Flughafen bestätigten wir ebenfalls diese Erscheinungen die aber meist irreversibel waren.

Es soll gleich betont werden dass sowohl nach Reintonbelastung (wie im ersten Fall) als auch nach Lärmelastung (wie im zweiten Fall) die Ermüdungssymptome schon beim gesunden Menschen bezüglich der Resultate der Einwirkungsdauer stark variieren d h sehr individuell zum Ausdruck kommen.

Die Erholungsdauer des Gehörorgans ist analog der Belastungsdauer beträgt also z B 24 Stunden wenn 3 bei 90 db mit 6 9000 Hz belastet wird (wie es Gampel und Hool festgestellt haben). Nach unseren Resultaten bewirken die Ultraschallbelastung den hohen Töne im Audiogramm als auch bei experimenteller Prüfung im hinteren Labyrinth eine reversible oder irreversible Veränderung bei Beschäftigten in der Umgebung von Düsenmotoren.

Es ist wohl bekannt dass im Laufe der Jahrzehnte bezüglich der hohen Tönen die Hörfähigkeit Stufenweise abnimmt. Das ist das physiologische Altern des Gehörorgans das mit 40 Jahren beginnt wobei noch nur 10000 Schwingungen pro sec gehört werden. Zwischen 50 bis 60 Jahren werden nur noch 10000 Schwingungen wahrgenommen und um das 70 Lebensjahr nur noch 8000 Schwingungen aufgefangen.

Die Einwirkung des Lärmes schädigt sowohl das periphere Organ (Innenohr Haarzellen Schädigung) als auch die akustischen Zentren. Dies ist schon bewiesen worden indem man nach Lärmelastung des Innenohres eine Schwerhörigkeit des anderen Ohres bemerkte.

Das Bodenpersonal zeigt besonders bei Düsenjägern ausser bei Schwerhörigkeit eine Neigung zum Schlaf. Es ist ja bekannt dass die monotonen Geräusche zum Schlaf Veranlagung geben und dass allgemein der Lärm die Arbeitskraft erschöpft.

Geräusche über 130 bis 140 db bewirken bei 20000 Hz Störungen indem sie durch Schwingungen auf dem Skelett und diese besonders schädigen. Wie bekannt die Prüfung der Motoren findet von Mechanikern im Vollauf der Maschine statt d h er steht unter einem Lärm von 140 db. Die Schädigung aussert

sich auch von Seiten des Fastgefühls des Sehorgans und in einer Schwäche der Gliedern besonders der Beimgelenke

Bei 170 db haben wir Erscheinungen von Seiten des autonomen Nervensystems (Schwindel, Nausea, Erbrechen, Pulsbeschleunigung oder Arrhythmie, Erhöhung des endolymphatischen Druckes und Blutdruckes, Spasmen, Coma)

Bei den Düsenmotoren sind die Infra-Schwingungen (unterhalb 16 Hz Energie) sehr gering, dagegen der Ultraschall sehr stark vorhanden. Letztere bewirken a) physiko-chemische und b) thermische Änderungen, weil a) die Zellen nekrotisieren können und b) die Keimzellen erzeugungs-fähig gestalten. Es wird behauptet, dass das Personal, das mit den Düsenmotoren beschäftigt ist, nichts zu fürchten hätte, weil angeblich die Turbinen nicht im Stande sind, über 120 db bei 22000 Hz oder 110 db bei 30000–40000 Hz zu erzeugen und weil die Haut je höher die Frequenz ist, um so weniger im Stande ist, die Schwingungen aufzunehmen. Zum Beispiel bei 400 Hz die Haut resorbiert 48%, Schwingungsenergie bei 6000 Hz 1% und bei 800 Hz 0,1%. Daneben muss noch die db-Zahl und die Dauer noch erörtert werden. Bei unseren Versuchen zeigten sich schon bei 20 db nach 1 Viertelstunde Belastung mit 20000 Hz Reinton folgende Erschöpfungsergebnisse:

Bei den Fliegern war dies bekannt, dass sie mit der Zeit eine Innenohrschwäche sowie eine Unterregbarkeit aufweisen. Bei ihnen handelte es sich um eine dauerhafte Schädigung infolge der langzeitigen Belastung durch Lärmeinwirkung. Dies lässt sich experimentell beweisen dadurch, dass die Vestibulärreaktion sowohl nach calorischer als auch nach rotatorischer Prüfung sich vermindert ausfällt. Hierzu haben wir das Vorbeizeigen Baranys und die Fallreaktion Rombergs untersucht. Es ergab sich, dass ersteres nicht immer in seinem typischen Form nachweisbar war, letztere dagegen fand sich unbeeinflusst. Dies deckt sich mit den empirischen Beobachtungen der Fliegerkandidatenuntersuchungskommission, welche wie bekannt besonders auf etwaiges Schwindelgefühl und Strabismusstörungen während der ersten Prüfungsflystunden für die Tauglichkeit der Kandidaten Wert legt und nicht auf die Ergebnisse der Vestibulärprüfungen.

Unsere Beobachtungen auf diesem Thema sind folgende. In 14 Personen mit normaler Hörfähigkeit und einer physiologischen Reaktion des hinteren Labyrinth wurde die Wirkung auf den Gehörsorgan in folgender Weise untersucht:

- 1) Mit tiefen Schallschwingungen
- 2) Mit Mittelschallschwingungen
- 3) Mit Ultraschallschwingungen

und wurde das Ergebnis dieser Wirkung auf dem vorderen und insbesondere auf dem hinteren Labyrinth geprüft. Die Ergebnisse sind folgende:

1) Die Wirkung der Schallschwingungen tiefen Tones auf dem Gehörsorgan von der Dauer 15–30 Minuten verursachen keine bemerkenswerten Störungen in dem vorderen und hinteren Labyrinth bei der experimenteller Prüfung.

- 2) Die Wirkung mit der Mittelschwingungen von der Dauer 15–30–40

Minuten verursachen zwar auf den vorderen Labyrinth keine bemerkenswerte Senkung des Gehörs aber auf dem hinteren Labyrinth verursachten Zunahme der Reflexerscheinungen des horizontalen Bogengangs die aber nicht die 5 Sekunden überschreitet bei der Experimenteller Prüfung

3) Die Wirkung der Ultraschallschwingungen von 20000 Hz verursacht Störungen sowohl auf dem Organ des Vernehmens des Schalles (Cochlearis) wie auch auf dem hinteren Labyrinth und wurde Experimentelle bewiesen Diese Störungen hängen ab a) von der Wirkungszeit des Ultraschalles b) von dem Zustand des Mittelohrs c) vom Alter So hatten wir

a) Bei Personen von einem Alter zwischen 20 und 25 mit physiologischer Fähigkeit des vorderen und hinteren Labyrinth wurden auf die Wirkung von Ultraschall 15 30 Minuten Die Hörbarkeit nach dieser Wirkung zeigte eine Senkung schwankend zwischen 10–25 db und betrifft hauptsächlich die hohen Töne Diese Senkung wurde auch nach 2 Stunden vorgefunden indem sie nach 24 Stunden auf das Physiologische zurückkam

Die Reaktion des hinteren Labyrinthes untersucht nach Kobrak (durch Drehung und laterale Reizung) zeigte eine Abnahme (Hypesthesie) d.h. während sie eine Reaktion nach 7–8 Drehungen zeigen sollte mit Dauer nachystagmus von 22–24 Sekunden zeigte sie nach der Wirkung von Ultraschallschwingungen einen Dauernystagmus von 15–16 Sekunden Diese Abnahme der Reaktion wurde nachgeprüft nach 2 Stunden und wurde 17 Sekunden gefunden Nach 25 Stunden kam es wieder zum Normalwert

Bei denselben Personen wurden Ultraschallschwingungen angewandt von einer Zeitdauer über 30–40 50–60 Minuten Dabei ging die Hypesthesie des hinteren Labyrinthes nicht unter 12 Sekunden Diese Personen wurden wieder nach 2 Stunden untersucht und zeigten eine Hypesthesie wie oben erwähnt Nach 24 Stunden kehrte die Reaktion des hinteren Labyrinthes wieder zum Physiologischen zurück bis zur Wirkung der Ultraschallschwingung von 60 Minuten

b) Bei noch 3 Patienten mit totalem Verlust des Trommelfelles zeigten die Ergebnisse ebenfalls nach Ultraschallschwingungen in einer Zeitdauer 15 und 30 Minuten eine grössere Senkung der Hörbarkeit und eine grössere Hypesthesie die länger dauert Bei diesen drei Personen kehrten die Ergebnisse nach einer tympanoplastischen Operation genau so wie bei den 14 vorigen Personen zurück

c) Bei noch 3 Personen von einem Alter von 55–60 Jahre war die Ultraschallwirkung auf das vordere Labyrinth grösser (grössere Senkung der Hörbarkeit) und auf das hintere Labyrinth eine Hypesthesie wie bei den ersten 14 Personen festzustellen

Ausser den letzten 6 waren die übrigen 14 Personen an denen die Versuche durchgeprüft wurden Studenten die sich freiwillig zur Verfügung stellten

Nach Hartigan zitiert vorübergehende Schwellenänderungen die nach 3 Minuten verschwinden Dies soll die Erholungssphäre – Hörermüdung sein Letztere ist unproportional wenn der Reiz n mehr als 20 db beträgt 20

Es ist also die kritische Schwelle. Dickson u. a. meint, dass die Gleichgewichtsstörungen bei den Piloten auf den Irm zurückzuführen sind. Connegheim betont den Zusammenhang zwischen vorderem und hinterem Labyrinth und hält sie als Reaction Vestibularis sonore. Er fand, dass bei 129 Patienten 72 diese Reaktion aufwiesen. Er konnte dieselbe auch bei Tauben hervorrufen. Levy und Montalcini sprechen von vestibulären akustischen Zentren bei Hunnern. Much und Camisarova fanden nach kalorischer Reizung nicht nur vestibuläre Erscheinungen, sondern gleichzeitig eine akustische Verminderung statt.

Unsere Aufgabe liegt darin, dass wir die eventuelle Schädigung durch Irmwirkung sowohl bei den Lufthafenpersonal und insbesondere bei den Dusenmotoren, beauftragten Bodenpersonal (Arbeiter, Angestellten) und den irgend wie konstant beschäftigten Personen dieses Betriebes zu prüfen. Um feststellen, ob und in wie weit der Irm und die Vibrationswellen die betreffenden schädigen und schliesslich, ob der angerichtete Schaden ein vorübergehender oder ein bleibender ist. Im Laufe dieser Untersuchungen nahmen wir stichprobenweise ebenfalls die Bewohner der Lufthafenegend unter Auge. Unsere Ergebnisse decken sich mit älteren Beobachtungen von anderen Seiten bezüglich statoracustischen und psychosomatischen Störungen, die durch Irm erzeugt wurden. Eine statoracustische Reaktion konnten wir auch experimentell nach Reinton-Belastung hervorrufen, wie schon erwähnt wurde.

Ausser der konstitutioneller Beschaffenheit des Gehörorgans (Cochlearisresistenz) müssen wir dabei noch mit der individuellen Konstitution rechnen.

Aus kommunalhygienischen Standpunkt Personen grosser Sensibilität gegen den Umweltirm zuzugewöhnen einen Reiz, der vermutlich sehr schädlich sein soll.

Personen, die in Irmreichen Betrieben beschäftigt sind, zeigen öfters und häufiger ausser der statoracustischen Störungen (Ohrgeräusche, Diplacusis, Schwerhörigkeit, Nausea, Schwindel, Gleichgewichtsstörungen, Unwillen, Sinnesstörungen, Angstzustände, Befalltheit, Überempfindlichkeit, psychokinetische Trägheit, Unlust, Kopfschmerzen, Kollapsus und andere dienecephalo-vegetativen Störungen) Funktionsanomalien im Vergleich zu den Beschäftigten in Irmfreien ruhigen Betrieben, denen die Abgabeleistung eine höhere ist. Es soll gleich hier betont werden, dass durch Irmstress beim Experiment die Raten eine Störung der endokrinen Drüsen zeigten (Sachler u. a.). Bei unseren Beobachtungspersonen war mit der Zeit die Arbeitsfähigkeit vermindert. Sie zeigten Kreislaufstörungen und Blutdruckstörungen, Pulsbeschleunigung und Arrhythmien, rasche Ermüdung, Anfang gestörten Schlaf, später Schlafsucht und andere psychosomatische Störungen. Von anderer Seite wurden schon in physicochemischen und thermischen Änderungen berichtet. Sowohl das Lufthafenpersonal durch die Barometerdruck, Leuchtigkeit und Temperaturveränderungen, noch dazu, als auch das Bodenpersonal zeigten, je nach Beschäftigung, bei den Dusenmaschinen eine grössere Erschöpfung.

Wie bekannt der Lärm zwischen 20–30 phons gilt als nicht störend während der von 50–60 phons als ertraglich gilt und derjenige von 80–95 phons als störend bezeichnet wird. Von da ab und darüber als unertraglich und schädlich einwirkt über 140 db wird er schmerzhaft empfunden selbstverständlich hängt es von der Dauer der Höhe (Frequenz) ab und ob er kontinuierlich oder in Intervallen sich wiederholt ob er im geschlossenen Raum oder im Freien und von der Entfernung der betreffenden Person ob letzterer geschützt oder ungeschützt ist vom alter psychischen und körperlichen Zustand ob der betreffende ruht oder beschäftigt ist und von den vorerwähnten individuellen Konstitutionsfaktoren. Unser Frachtens auf alle diese Faktoren beruhen die Verschiedenheit der statistischen Angaben die schon 800 3000 Stunden im Fluge halten. Beim Fliegerpersonal bezüglich statonkustischen Störungen zeigte sich beim 2/3 ein Hörverlust von 10 db und beim 1/3 10–20 db bezüglich der Luftleitung. Weniger betroffen zeigt sich die Knochenleitung. Das Sprachgehör wird 80/100 beeinträchtigt. Die Vestibulusreaktion war dreimal erhöht sonst vermindert.

Sackler und Mitarbeiter in der *Acta Endocrin* 31 (1959) Nr 3–400 stellten Lärmschäden an den Endocrinen Drüsen experimentell fest. Für weitere Beschreibungen siehe Presse Berichte. Von W. Furrer wurden die Eigenschaften des Dusenalarms physikalisch analysiert. Er fand eine Komponente von 800 Hz die besonders stark ausgeprägt sein soll in den sie um 70 db über das allgemein Geräuschspektrum herausragt.

Vallancien beschrieb dass eine Einwirkung von Ultraschall 60 sek lang den Tod bestimmter Rassen von weissen Mäusen durch Herzstillstand herbeiführen kann. Wird die Schalleinwirkung unterbrochen dann kann man durch Herzmassage die Tiere wiederbeleben. Die Mäuse drehen sich wie toll um ihre eigene Achse dabei. Die Kühe legen weniger Milch in der Gegend wo Dusenmaschinen fliegen. Die Hühner legen schalllose Eier. Mehrere hunderte von Wiesel starben durch die Einwirkung von Lärm der Dusenmaschinen in Schweden. Es hiess dass die durch Schreck eingingen. Unsere Frachtens stellen oben erwähnte Beispiele die Bestätigung der tierexperimentellen Befunde Vallanciens dar.

Nach Dr. Angeluschoff geschieht die Vibration auf die Zellen nicht über die Intensität sondern über die Frequenz letztere bringt Zellstrukturen zur Resonanz und bewirkt dadurch chemische Veränderungen in den Proteinen mit Ionenwanderungen. Eine gewisse Rolle spielt dabei das Kalium als der bedeutungsvollste Elektrolyt für die Überleitung von Reizen.

Ultraschall führt zur Verarmung von Kalium in den nervösen Substanzen dadurch lässt die Reaktion auf Vibrationen nach und es resultiert dadurch Schwerhörigkeit und unseres Frachtens nach gleichzeitig eine Unterregbarkeit des hinteren Labyrinthes. Casarati konnte nach 10 Minuten Dauer Ultraschalleinwirkung eine Auflöserung der Perilymphe hervorrufen. Im übrigen ist es wohl seit langen bekannt dass die Ultraschalleinwirkungen einerseits physikalisch-chemische Änderungen und im Sinne einer Nekrose der Zellen als auch in tiefe thermische Änderungen hervorrufen und verursachen.

können. Ebenfalls wurde eine Erhöhung der Polymorphie und der Isomorphie festgestellt.

Unser Irrtumszusammenhang der aufgefundenen Schwingungen gilt es noch andere da wir bekannt bei diesen Flugzeugen Schwingungen bis 40 000 Hz vorfinden können. Die aber wegen der Minderwertigkeit der im Handel stehenden Apparate und insbesondere der Mangelhaftigkeit deren Mikrophone (wobei der Schall über 16 000 Hz überhaupt nicht aufgenommen wird um von Schallmeter registriert zu werden) über Tonbandübertragung zu Oscillographen sind noch mangelhaften deswegen Personen die den diesen Flugzeugen ausgesetzt sind beschreiben denselben als viel unangenehmer als diejenigen der Kolbenflugzeuge (siehe Diagramm Nr. 5) da die Störungen während des Gespräches aufzeichnen da wie bekannt der Lärm der Jet weiter ausbreitet wird einerseits und andererseits durch das Vorhandensein der Ultraschall Schwingungen bereichert wird.

Bei den Zivilflugzeugen werden die Maschinen zirka 5 Minuten lang auf dem Boden vor den Flug vorbereitet und ausprobiert. Erst dann können sie starten. Der Lärm beträgt dabei 110–140 db in der Nähe der Motoren und in der Höhe von 100 m ist die Stärke des Lärmes zirka 90 db. Der Reihe nach ist die Caravelle weit lauter als Boeing 707 und zuletzt der Comet 4B. Die Kolbenpropellerflugzeuge weisen als charakteristischem einen tiefen Grundton um 100 Hz auf plus harmonische Töne die eine schwankende Stärke um 120 db aufweisen. Der erzeugte Lärm ist nicht kontinuierlich.

Bei den Jet Maschinen ist der Lärm dagegen kontinuierlich und gleichmäßig und zeigt als Charakteristischem eine Frequenz von zirka 200 Hz und eine Stärke um 130 db. Der Lärm wird viel breiter verbreitet in Vergleich zu den vorigen Flugzeugtypen. Die Maschinen müssen im Vollauf vom Mechaniker geprüft werden d. h. der betreffende unterliegt einem Lärm von 140 db.

Es heißt dass der Lärm subjektiv zuerst als unangenehme Empfindung wahrgenommen wird und erst dann kann derselbe krankheitszustände auslösen oder begünstigen (Schneider u. a.). Auf Grund aber unserer Beobachtungen meinen wir dass die unangenehme Empfindung und die Schädigung Hand in Hand vor sich geht ja bei Ultraschalleinwirkung kommen Störungen ohne markante unangenehme Empfindungen vor. Da liegt die Gefahr der Düsenmotoren.

Die Bilder und Schemata stammen aus der Doktorarbeit Herrn Kousoulakos die unlängst aus meiner Klinik erschien. Dr. Kousoulakos hat viele seiner Beobachtungen während seiner Tätigkeit im NATO gesammelt.

Eine Anzahl von Beobachtungspersonen zeigt der Reihe nach (auf 100) folgende Störungen:

Unlust Abgeschlagenheit (somatisch und geistig) Sinnesstörungen Müdigkeit Gedächtnisschwäche Kopfschmerzen Schlafsucht anfangs Schlaflosigkeit Schlafstörungen Nausea Anchos Drehschwindel Sensationen an den Beinen Blässe Nackenschmerzen Gleichgewichtsstörungen Erlernen Schwere Sausbrüche Angstgefühl Appetitlosigkeit Befangenheit Unentschiedenheit

denheit Tachycardie Extrasystolen Psychische Labilität und Blutdruckschwankungen

Diese Störungen treten meist gehäuft oder auch vereinzelt vor und zeigen sich oft nach Lärmeinwirkungen und stärker bei überarbeiteten übermüdeten Personen. Kleine Kinder, die zum ersten Mal dem Dusenmaschinenlärm ausgesetzt sind, beginnen zu weinen, sogar während des Schlafes. Eine Anzahl von denen zeigen Zuckungen, die sicher eine Belastung des Nervensystems beweisen. Über Spätfolgen wäre es noch vorzeitig zu berichten. Die Zahlen sind noch zu gering, dass man statistische Schlüsse ziehen dürfte. Kinder, die ständige Bewohner der Flughafengegend sind, zeigen erhöhte Nervositätserscheinungen (leicht reizbar, zappelig) und diencephalovegetative Störungen, da wie bekannt, das nervovegetative System bei den Kindern stark entwickelt ist.

Wir haben uns durch folgenden Apparaturen und Maschinen bedient:

- 1) Ichometer (siehe Abbildung Diagramme)
- 2) Fosmatoskop zur Analysierung der Töne
- 3) Typen von Kolben Propeller Aeroplanen
- 4) Viscount (Dusen Propeller Flugmaschinen)
- 5) Jet Dusen Flugzeuge
- 6) Caravelle Boeing Comet B4

7) Abbildung von erwähnten Flugzeuge des I Arms beim Starten, wobei die Maschine mit 90% Energie arbeitet

8) Diagramm des vorgerufenen Lärmes von einem Dusen und einem Kolben Flugzeug im Fluge und deren Einwirkung während einer Sprachunterhaltung und deren Verständigung, d. h. beide bewirken denselben Masking Effekt

9) Diagramm einer Jet Maschine und einer Kolbenmaschine beim Starten, wobei der Lärm, der durch die erste Dusen Maschine erzeugt wird, das Sprachverständnis vollkommen verhindert, dagegen durch den Lärm der zweiten Kolbenmaschine das Gespräch (das Vorgesprochene noch hörbar ist) in der Nähe sich befindenden Personen

10) Die Einwirkung auf den I Arm im Luftschiff mit und ohne Kompression

11) Hervorgerufenener I Arm bei abnehmen eines Dusen und eines Kolben Flugzeuges

12) Diagramm als Beweis

Die Dusenmotoren erreichen eine Schwingungszahl bis 40000 Hz. Der Ichometerapparat hört nur bis 10000 Hz, maximum 16000 Hz, dadurch ist die Diskrepanz zwischen unangenehmen Empfindungen und der db Stärkeaufschreibung zwischen den verschiedenen Luftschiffentypen zu deuten. Wir sind vorläufig nicht im Stande, die db, die durch Ultraschallschwingungen erzeugt werden, zu registrieren, wir sind aber im Stande, die unangenehme Erscheinungen zu erfassen und zu hervorheben und leider nur deren Schädigungen zu konstatieren.

INHIBITORY EFFECT OF STERILE OTITIS MEDIA EXUDATES ON THE CYTOPATHOGENICITY OF HERPES SIMPLEX POLIOMYELITIS AND ADENOVIRUSES IN HELA CELLS

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University of Helsinki*

The inhibitory effect of bacteriologically sterile otitis media exudates on multiplication of three types of viruses was studied. Most of the exudates tested were found to have an inhibitory effect on cytopathogenicity of these viruses in HeLa cell cultures.

In teamwork with several co-workers it has been possible earlier to demonstrate that the intratympanic exudate in acute otitis media is active against bacteria and viruses (4, 5, 6).

This report describes further studies on the nature of the antiviral activity of sterile middle ear exudates.

Material and Methods

Otitis media exudates

About 100 sterile samples of otitis media exudate were collected. Specimens of the exudate were taken with a 2 ml syringe and a long, bayonet shaped needle through the perforated drum membrane. The specimens were stored at -20°C in small glass tubes with rubber stoppers. Of these specimens 20 were selected which contained no blood and were sterile according to bacteriological tests.

From some of the patients a blood specimen was taken at the same time as the exudate. The sera were stored in the same way as the exudates.

Virus strains

The following virus strains were used: Struett strain of poliomyelitis type 3; prototype strain of adenovirus type 6; human kidney passage of a measles virus isolate (1) and the herpes simplex virus which was our own isolate in HeLa cells. The herpes simplex, measles and adenoviruses were stored at -60°C , the poliomyelitis virus at -25°C .

Accepted by grants from the Sigrid Juselius Foundation

Cell cultures

HeLa cell cultures were grown in tubes in a medium consisting of 40% human serum and 60% Hanks solution. At the time of inoculation the tubes were washed three times with Hanks solution and 1.0 ml of a medium consisting of 5% horse serum, 20% tryptose phosphate and 75% medium No. 199 was added.

Neutralization tests

Equal parts of 1/10 diluted virus and undiluted otitis media exudate or serum were mixed. After one hour's incubation at 37°C serial tenfold dilutions both of the mixtures and of the virus controls were prepared in the cell culture maintenance medium. From each dilution 0.1 ml was inoculated into each of three cell culture tubes which were incubated for 4-7 days at 37°C. The cultures were observed for cytopathogenic effects at intervals of 1 to 3 days. Infectivity titers were calculated by the Reed-Muench method (3) and expressed as the number of tissue culture infectious doses (TCID₅₀) per 0.1 ml.

In the preliminary experiments no significant differences could be noted in the infectivity titers of virus controls mixed in equal parts with undiluted rabbit serum or with cell culture maintenance medium before incubation at 37°C. Therefore the virus controls were mixed with an equal volume of maintenance medium.

To assess the reproducibility of the test two identical experiments with herpes simplex virus mixed with an exudate pool were made on different days on different lots of HeLa cells. The difference in the titers of these two separate tests after observation periods of 2, 3, 4 and 5 days was 0.5 log or less.

Complement fixation tests

Complement fixation tests were performed by the Bengtson technique with some modifications (2): two full units of complement were used. The complement fixation antigens were prepared in HeLa cells (herpes simplex) and in a continuous line of human amnion cells (measles).

Results

Inhibitory effect of exudate pools

The first series of experiments was made with the pooled exudates of five patients. This same pool was tested with herpes simplex and adenovirus. Two days after the inoculation the infectivity titer of the herpes simplex virus mixed with the exudate pool was 2.5 log units lower than the titer of the control virus (Fig. 1). Later the difference in titer was smaller and on the sixth day when the final examination of the tubes was made it was only one log unit.

The inhibitory effect of this exudate pool on the cytopathogenicity of the adenovirus was even greater. After two days the titer of the virus mixed with the exudate pool was at least 4.5 log units lower than the control and after 4 days 4 log units lower.

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Material and Method

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About 100 sterile samples of otitis media exudate were collected. Specimens of the exudate were taken with a 2 ml syringe and a long 19-gauge shaped needle through the imperforate drum membrane. The specimens were stored at -20°C in small glass tubes with rubber stoppers. Of these specimens 30 were selected which contained no blood and were sterile according to bacterial tests.

From some of the patients a blood specimen was taken at the same time as the exudate. The sera were stored in the same way as the exudates.

Virus strains

The following virus strains were used: Suckoff strain of poliomyelitis type 3; prototype strain of adenovirus type 6; human kidney passage of a measles virus isolate (1) and the herpes simplex virus which was our own isolate in HeLa cells. The herpes simplex, measles and adenoviruses were stored at -60°C , the poliomyelitis virus at -25°C .

Advised by grants from the Sigrid Juselius Foundation

specimens had poliomyelitis type 3 neutralizing antibodies and all four exudates had an inhibitory effect on the cytopathogenicity of this poliomyelitis virus. In three of the tests the titers of the virus mixed with the exudate and with the serum were the same. In the fourth the neutralizing activity of the serum was 1.5 log units greater than the inhibitory activity of the otitis media exudate.

Herpes simplex and measles complement fixing antibodies in individual exudates and sera of the same patients

Herpes simplex and measles complement fixing antibodies could be found in some of the exudates tested (Table 1). The exudates only contained CF antibodies in those cases in which the corresponding serum contained CF antibodies against the same virus and vice versa. The CF antibody titer in the exudate was the same as or half that of the corresponding serum.

CONCLUSIONS

It was found that otitis media exudates have an inhibitory effect on the cytopathogenicity of herpes simplex, poliomyelitis and adenoviruses in HeLa cells. This inhibitory activity of the exudates could be identified as due to virus neutralizing antibodies because

- (a) it was not inactivated at 56°C in 30 minutes
- (b) in most patients the inhibitory activity of the exudates and the virus neutralizing activity of the serum were of the same level
- (c) the exudates had specific complement fixing virus (herpes simplex and measles) antibodies

Together with the antibacterial effect earlier demonstrated in the middle ear exudate, virus neutralizing antibodies probably protect the ear against infection in most cases of nasal and pharyngeal inflammation.

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University Clinic of Helsinki

DISCUSSION

H. A. F. van Dishoeck I agree with Surala that virus infection must play an important role in catarrhal otitis media. We often found many lymphocytes and only few leucocytes in the exudate as in benign viral meningitis which is often the cause of sudden perceptive deafness. In our virological department Prof. Verlinden was able to culture viruses from some of these exudates.

In our opinion viral infection often precedes a bacterial infection. An example is the purulent discharge following a common cold. Also for the grippe this mechanism is well known. The virus damages the epithelium and opens the tissues for the bacterial invasion.

HISTOPATHOLOGY IN MÉNIÈRE'S DISEASE

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Besides the well known and established pressure changes in the endolymphatic system pronounced degenerative changes of the peripheral acoustic system can be demonstrated

As pointed out by Williams & Altmann in their comprehensive reviews of Ménière's disease by Hallpike & Dix and Cawthorne & Mittermaier, the clinical picture of non infective aural gyratory vertigo plus tinnitus and deafness as originally described by Ménière in 1861 has been misunderstood and misquoted to such an extent that much confusion has arisen. According to Ménière's original descriptions, it is beyond doubt that he was the first to stress that the patients had a disease in the labyrinth and not in the brain. He came to this conclusion by relating Flourens' results after experiments on the semicircular canals of pigeons to careful clinical observation and examination of a large number of patients.

Ménière mentions in one of his articles two colleagues suffering from attacks of vertigo deafness nausea and vomiting accompanied by fear because they thought they had a disease of the brain. In this connection he points out the importance of performing acoustic tests. In his first paper he stresses that

(1) A completely healthy internal ear can suddenly become the seat of disturbances consisting of tinnitus accompanied by more or less pronounced hearing loss.

(2) The disease formerly considered cerebral is localized to the internal ear and gives vertigo uncertain gait nausea and vomiting besides deafness.

(3) The disease which can be intermittent is followed by progressive hardness of hearing or complete deafness.

From the same paper may be quoted: *Nous croyons que les recherches microscopiques dirigées dans ce sens viendront à l'appui de notre opinion et contribueront à jeter du jour sur la nature de certaines cophoses. Et nous ajoutons que tout ce qui peut conduire au diagnostic exact d'une maladie, et par conséquent à établir son degré de curabilité nous semble un service rendu à la science.*

Ménière not only paid attention to the importance of examining the hearing but also made a plea for histological examination of temporal bones from such cases. This was done in 1938 by Hallpike & Cairns. The main points added to this clinical picture in the past century have been the fluctuation of hearing (Crowe and McGinley & Dederding) and the recruitment phenomenon (Hallpike Dix & Hood). Vestibular tests were not mentioned by Ménière for natural rea-

DISCUSSION

H. A. I. van Dishoeck I agree with Shirata that virus infection must play an important role in catarrhal otitis media. We often found many lymphocytes and only few leucocytes in the exudate as in benign viral meningitis, which is often the cause of sudden perceptive deafness. In our virological department Prof. Verlinden was able to culture viruses from some of these exudates.

In our opinion viral infection often precedes a bacterial infection. An example is the purulent discharge following a common cold. Also for the grippe this mechanism is well known. The virus damages the epithelium and opens the tissues for the bacterial invasion.

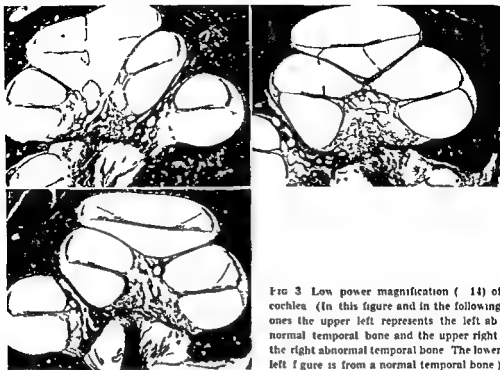


FIG 3 Low power magnification (14) of cochlea (In this figure and in the following ones the upper left represents the left ab normal temporal bone and the upper right the right abnormal temporal bone The lower left figure is from a normal temporal bone)

ness have been regarded as Meniere's disease and no proper distinction was made between otitis with labyrinth oedema, occlusion of the Eustachian tube, otosclerosis, brain tumors and in particular cases of neuroma of the eighth nerve (Frankl-Hochwarth). Similarly Magind & Dederding have maintained that

(1) cases of middle ear suppuration with collateral labyrinth oedema may be treated as Meniere's disease from which they do not in reality differ

(2) cases of middle ear catarrh and tubal stenosis are subject to the same giddiness and nystagmus as Meniere patients

(3) that apparently typical otosclerosis cases with a negative Rinne and very severe bass deafness may turn out to be Meniere's disease as shown by improvement when treated with diuretics and

(4) that apparently typical Meniere's disease may accompany or initiate a series of intracranial affections (for example tumor of the acoustic nerve)

On the other hand it is also known that some cases of Meniere's disease have had to be operated on for acoustic neuroma or have been successfully treated with stapes mobilization

When so much confusion exists about the clinical picture it is no wonder that the pathological changes in the temporal bones from such patients may be rather confusing too

Only a few histopathological textbooks describe the pathology of Meniere's disease. In *Handbuch der Speziellen Pathologischen Anatomie und Histologie* by Henke & Lubarsch only cases with bleeding of the labyrinth are men-

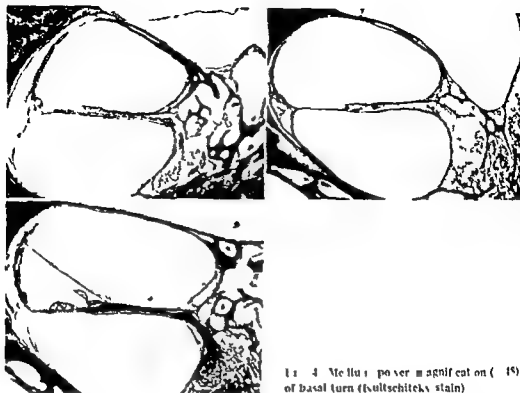


Fig. 4. Melius, power magnification (15) of basal turn (Kultschitzky's stain)

tioned often in combination with leucemia. *The Histo Pathology of Ear, Nose & Throat* by Igelson & Wolff mentions Meniere's syndrome under the heading of hydrops and writes: "A toxic chemical change in the surrounding lymph fluids evidently has a solvent effect upon the membrane so that it disintegrates. No fragments of cells of the organ of Corti are seen, so complete is the chemical resorption. An identical pathological process occurs in the vestibular portion of the labyrinth. Cristae and maculae may become completely atrophic as may the ganglion cells of the eighth nerve." In *Oto Laryngic Pathology* by Ash & Raum (Armed Forces Institute of Pathology, 1956) you find some excellent photographs of a case of Lindsay with marked dilatation of the cochlear duct and dilated saccule and utricle. Furthermore it says: "Other anatomic changes are lacking and there is no evidence of an inflammatory factor. The same pathological changes have been found in cases where there was deafness but no vertigo of the complete Meniere's syndrome accounting for this by assuming that the vertigo is an early symptom which may disappear at later stages."

Against this background one feels that Altmann and Wustrow & Borowski are justified in questioning the diagnosis in some histologically examined cases. One is likely to agree with Altmann's and Hallpike's statements concerning Wittmaack's case that this cannot be a case of Meniere's disease and the opportunity is taken to demonstrate a case of a type similar to Wittmaack's with a neuroma within the scala tympani but without dilatation of the endolymphatic apparatus and without Meniere's symptoms (Fig. 1).

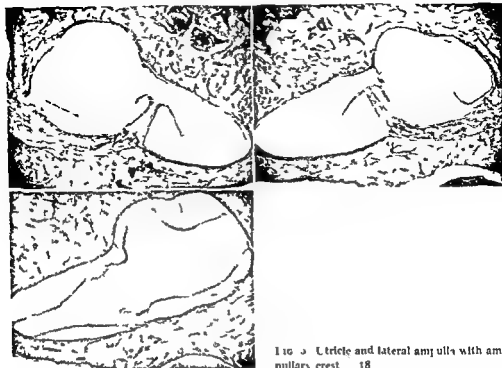


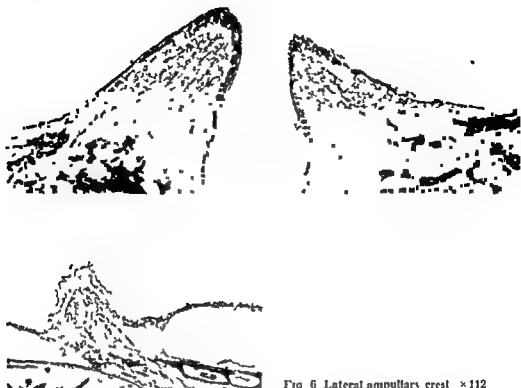
Fig. 3. Utricle and lateral ampulla with ampullary crest 18

When Wustrow & Borkowsky report four cases of Meniere's disease without endolymphatic dilatation it is probably because these were not cases of Meniere's disease. It is more difficult to account for the four reported cases of endolymphatic dilatation in cases which were not Meniere's disease. Wustrow & Borkowsky tried to explain this as an histological artefact. Theoretically, however, there should rather be a tendency to collapse of the endolymphatic system in particular due to the celloidin embedding.

In fact, when we went through our series of 120 human temporal bones from the last four years we found 18 instances of so called collapsed Reissner's membrane and in five of these adhesion of the utricular membrane to the macula. We did not find endolymphatic dilatation except in the following case.

The patient was a female born in 1899. Her troubles began in the summer of 1912, where suddenly she had an attack of violent gyrotory vertigo. Since then the patient had had repeated attacks of vertigo accompanied by a feeling of fullness in the head along with tinnitus and fluctuating hearing loss of bass type, leading to complete loss of hearing in the right ear and to pronounced loss of hearing in the left ear (Fig. 2).

On a cold caloric test performed in 1949 there was no response from the right labyrinth. On account of violent pelvic pains she was admitted to the neurosurgical department in 1954 and 1955. Here it was noted that the patient was practically deaf except from small remnants of hearing on the left ear. There had been no vertigo the last 10 years. She died in November 1955 from a pelvic tumor of unknown origin.

FIG 6 Lateral ampullary crest $\times 112$

The temporal bones were removed and fixed in 10% formalin. They were rather atrophic so that the anterior vertical canal was seen through the semi-transparent bone. They were decalcified in a buffer solution of sodium formate and formic acid, embedded in celloidin and cut in horizontal sections.

On histological examination pneumatization of the temporal bones was found normal. The labyrinth capsule showed no abnormality and particularly there was no sign of otosclerosis. The external auditory meatus was normal and so was the tympanic cavity regarding the mucous membranes and the ossicles.

In order to make comparison easier the sections of the abnormal temporal bones are shown along with sections from a normal temporal bone.

Low power magnification of the cochlea (Fig. 3) shows enormous dilatation of the scala media with obliteration of the perilymphatic space and displacement of Reisner's membrane into the apical portion of the scala tympani. In the right temporal bone the organ of Corti has disappeared completely. In the left temporal bone one can see a pronounced degeneration of the organ of Corti, most pronounced in the apical turn, something that might correspond to the low tone deafness in the initial stages of the disease.

At a higher magnification (Fig. 4) is seen a pronounced degeneration of the nerve cells in the spiral ganglion and of the nerves passing to the spiral ganglion. Perhaps there is some degeneration of the stria vascularis. Finally it may be noted that in the section from the right temporal bone there are practically no nuclei in the limbus spiralis of the upper turn.

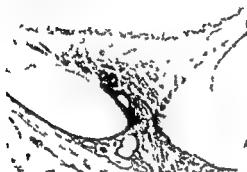


FIG 7 Macula of the utricle 48

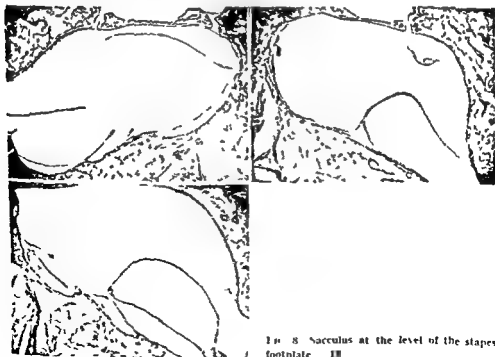
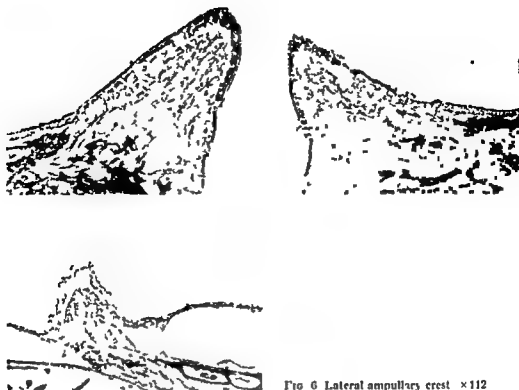


FIG 8 Sacculus at the level of the stapes footplate 49

FIG. 6. Lateral ampullary crest $\times 112$

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On histological examination pneumatization of the temporal bones was found normal. The labyrinth capsule showed no abnormality and particularly there was no sign of otosclerosis. The external auditory meatus was normal and so was the tympanic cavity regarding the mucous membranes and the ossicles.

In order to make comparison easier the sections of the abnormal temporal bones are shown along with sections from a normal temporal bone.

Low power magnification of the cochlea (Fig. 3) shows enormous dilatation of the scala media with obliteration of the perilymphatic space and displacement of Reissner's membrane into the apical portion of the scala tympani. In the right temporal bone the organ of Corti has disappeared completely. In the left temporal bone one can see a pronounced degeneration of the organ of Corti, most pronounced in the apical turn, something that might correspond to the low tone deafness in the initial stages of the disease.

At a higher magnification (Fig. 4) is seen a pronounced degeneration of the nerve cells in the spiral ganglion and of the nerves passing to the spiral ganglion. Perhaps there is some degeneration of the stria vascularis. Finally it may be noted that in the section from the right temporal bone there are practically no nuclei in the limbus spiralis of the upper turn.



FIG. Macula of the utricle 48

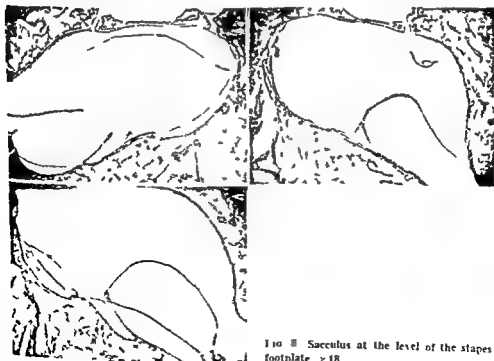


FIG. Sacculus at the level of the stapes footplate 48

hearing was described by Fournier Mittermaler and Bocca in Ménière's disease and in some cases of hearing loss caused by pathological changes within the cochlea or the cochlear nerve itself. Similar symptoms of vestibular hearing I could state in three cases of Ménière's disease, which revealed unilateral hardness of hearing to the 64, 125 and 250 cps tones, reduction of high frequencies from 1000 cps upwards, a positive recruitment phenomenon. The caloric test (44°C) was slightly diminished. In these cases good perception of the lower frequencies through the bone conduction was evident when putting the tuning forks C4, 125 and 250 to the mastoid bone. The patients were able to discern between perception of the tone and feeling of vibrations through the bone conduction, while the curve of the former superposed the bone conduction audiogram. Changeable values of 15-35 db were observed in the latter but in order to exclude the contralateral perception of the tested tones masking of the opposite ear was continuously applied. Conversely a normal auditory threshold was seen in two subsequent cases of Ménière's disease in which the caloric test was considerably diminished for which reason the better hearing of the lower tones through the bone conduction was not demonstrable at all. It happens thus because perception of the lower tones of a more specific vibratory character through the bone conduction is very probably mediated by the vestibulo-cochlear endings in question as was discussed by J. Bocca.

II. Tren et. Bei der Deutung des Morbus Ménièrei sui generis scheint mir die stärkere Berücksichtigung der Richtung des Nystagmus im Anfall und zwar bei der Beobachtung innerhalb der ersten 10 Minuten nach Anfallsbeginn ratsam zu sein. Bei strenger Definition des einseitigen Morbus Ménière haben wir gefunden, dass der Nystagmus überwiegend zur kranken Seite schlägt, gelegentlich zur Gesunden und in einer dritten Gruppe regellos mit dem Vorkommen auch von vertikalem Nystagmus. Es ist schwer vorstellbar, dass der oft massive Nystagmus zur kranken Seite ein Reizsymptom ist, so dass man an lymphokinetische Vorgänge infolge profuser Produktion von Labyrinthflüssigkeit denken muss. Nur der Nystagmus zur gesunden Seite konnte sowohl durch einen Gefassspasmus als auch durch Kompression der ihr zugehörigen Gefäße infolge endolabyrinthärer Drucksteigerung oder durch einen „sludge“ erklärt werden.

Zur Nomenklatur der Vestibularisstörungen meine ich, dass man ohne die Benutzung eines Formenkreises Ménière artiger Krankheitsbilder nicht auskommt, obwohl es sehr wünschenswert wäre, den Namen Ménières nur in Verbindung mit dem Morbus Ménière zu gebrauchen. In einem Schema der Vestibularisstörungen, das sich diagnostisch bewahrt hat, habe ich aus der Gesamtheit der Vestibularisstörungen die drei Gruppen des Morbus Ménière, des einseitigen plötzlichen Vestibularisausfalles und der Vestibularisstörungen mit Lage- und Lagerungs-Nystagmus abgegrenzt und als Nachbargruppe des Morbus Ménière den Formenkreis Ménière artiger Krankheitsbilder mit fließenden Übergängen zu den restlichen Vestibularisstörungen anderer Art eingefügt. Man benötigt den Formenkreis, um etwa doppelseitige oder rein vestibuläre Ménière sche Krankheiten oder Erkrankungen des ZNS mit symptomatischem Vestibularisanfallschwindel einordnen zu können, bis der weitere Verlauf zu einer Klärung geführt hat.

III. L. Wullstein. Der Nachweis des Hydrops ist uns auch beim Lebenden gelungen, was ich in Ergänzung zu dem Vortrage von Herrn Kristensen erwähnen möchte. Zusammen mit S. Rauch haben wir gelegentlich der Ménière Operation nach Cawthorne bei temporärer Eröffnung des Vestibulums den Inhalt zur chemischen Analyse

der Elektrolyte gewonnen. Wir wissen, wie gross der Unterschied an Kalium und Natrium in der Perilymphe bez. Endolympe ist. Bei einer Serie von Ménière Operationen ergaben sich bei sorgfältiger Entnahme in verschiedenen Portionen nur Elektrolyt Werte, die typisch für die Endolympe sind, dagegen konnten keine zur die Perilymphe typischen Analysen festgestellt werden (bis auf einen Fall mit wahrscheinlich kleiner Beimengung). Im Gegensatz zum normalen Ohre und von anderen Erkrankungen war es also nicht möglich gewesen zuerst die Perilymphe zu aspirieren und darauf das Endolymphsystem zu punktieren, weil der Endolymphraum bei diesen Spätstadien der Ménière-Erkrankung das Vestibulum fast vollständig ausgefüllt hatte.

G. Kelemen The excellent pathohistological analysis of Prof. Kristensen is a welcome addition to the relatively scarce pathological documentation of these conditions. There is microscopical evidence (demonstration of slides) that hemorrhage coming from the perilymph destroys the cupula. *Every erythrocyte coming in contact with the cupula acts as a specific poison.* As a crista devoid of its cupula cannot work and as the cupula does not degenerate, the consequences of these findings are obvious.

E. Bocca I have been prompted to enter this discussion by the interlocution of Dr. Laskiewicz, who has once more drawn attention to the fascinating problem of vestibular hearing in man. After the paper of last year in Vienna we carried out further research and we can fully confirm what Dr. Laskiewicz says, that is also in Ménière cases. Bone conduction for the low tones is preserved when the vestibular excitability is preserved and vice versa. I cannot say if this so called vestibular hearing is connected with the function of the vestibulo-cochlear fibers or with the sacculus only.

The only thing I can say is that among so many studies upon nystagmus etc., the study of this acoustic symptomatology of Ménière's disease deserves further and more extended research.

R. Mittermaier Zum Fragenkomplex der Ménière'schen Krankheit möchte ich zwei Bemerkungen machen.

1. Gibt es bei der Ménière'schen Krankheit wirklich ein besseres Hören durch Knochenleitung als durch Luftleitung im Bereich der tiefen Töne?

Hallpike hat solche Befunde ganz einfach als falschen Rinne bezeichnet. Aubry schreibt in seinem Lehrbuch die Knochenleitung könne in diesem Bereich gegenüber der Luftleitung etwas erhöht sein.

Zunächst wird man sich zu fragen haben, ob der Patient sich nicht irt und in Wirklichkeit den Ton nur auf dem besserhörenden Ohr wahrnimmt. Wir haben den Rinne'schen Wasserfüllungsversuch des Gehörganges auf der kranken Seite ausgeführt und dadurch festgestellt, dass nicht lediglich ein Hinüberhören auf das gesunde Ohr vorgelegen hat. Weiter ist zu fragen, ob der Patient die Töne durch Knochenleitung wirklich hört oder vielleicht nur fühlt.

Es gibt Patienten, die angeben, den Ton wirklich zu hören; bei den meisten wird es allerdings nur ein Fühlen sein (über den Wasserfüllungsversuch bei Ménière'scher Krankheit s. B. MITTERMAIER Über die Ménière'sche Krankheit HNO Bd 7, II 8 S. 22) (1959).

2. Einen Nystagmus zur gesunden Seite während des Anfalles wird man verhältnismässig einfach als Ausfallsnystagmus erklären.

Wesentlich schwieriger dagegen ist das gleichzeitige Vorkommen eines Funktions-

ausfallen im cochlearen Teil, also eine Taubheit, und eines Reizzustandes im vestibulären Bereich zu verstehen

Schilderung eines Falles von plotzlicher Ertaubung rechts bei tagelang anhaltendem Spontannystagmus nach rechts. Der Zustand besserte sich auf wiederholte sympathicusblockierende Novocain Injektionen. Es hat sich zweifellos in diesem Falle nicht um eine Menière'sche Krankheit sondern mit aller Wahrscheinlichkeit um eine spastische Störung gehandelt. Wie ist das Zusammentreffen von Ertaubung mit lange anhaltendem Nystagmus zur ertaubten Seite zu erklären?

P. F. Hennebert L'examen audiométrique des cas de maladie de Ménière par des méthodes utilisant le balayage de fréquence montre que les altérations de l'audition sont toujours bilatérales contrairement à l'opinion classique qui considère la maladie de Ménière comme une atteinte unilatérale du labyrinthe

H. A. Kristensen (Reply) I like to thank the discussers for their interest in my paper

To Professor Laskiewicz who as far as I understood, asked about changes histologically in the central connections of the eight nerve, I would like to say that with special staining of the intratemporal part of the eight nerve no changes could be demonstrated. I should have liked to have the oblongate medullary and the brain examined too but we missed that chance due to various circumstances

Zu Herrn Professor Jenzel wurde ich gern sagen dass wir es sehr wichtig finden zwischen Mb. Ménière und andere vestibularische Erscheinungen zu sondern zu versuchen obwohl es wohl logisch wäre, ob es Verbindung zwischen verschiedene vestibuläre Störungen bestehe. Dies haben wir jedoch nicht histologisch untersuchen können. Auch wir finden dass in akuten Fällen von Mb. Ménière Nystagmus nach der erkrankten Seite schlägt und ich stelle mich vor, dass es sich um eine Stimulation drehe

Ich danke Herrn Professor Wullstein für seine Mitteilung über Untersuchungen des Natrium- und Kaliumgehaltes des Labyrinthes wo seine Befunde auch auf endolymphatische Dilatation deuten

To Professor Kelemen I would like to say that one of my assistants is also working with P.A.S. staining of temporal bones from cases of diabetes where he finds pronounced changes of the vessels plus hemorrhage, but in the case reported he found no such changes on P.A.S. staining

Zu den Herren Professoren Bocca und Mittermaier möchte ich sagen dass wir nie verlängerten Knochenleitung gefunden habe und speziell nicht in dem referierten Fall

A Professeur Hennebert je veux dire que nous avons eu les mêmes expériences que mes maîtres les docteurs Cawthorne et Hallpike, que les alterations sont pour la plupart unilatérales dans la maladie de Ménière, mais que la maladie après une durée longue peut être bilatérale aussi

ÜBER DIE EIGENDÄMPFUNG DER STIMMBÄNDER BEIM SCHWINGUNGSVORGANG

JOSEF IESCHKE
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Es wird nachzuweisen versucht, dass die charakteristische unilaterale und vorwiegend horizontale Schwingungsart der Stimmbänder durch die vom Verfasser beschriebene antagonistische Zugelwirkung der Muskelfasern vom Internus und Externus verursacht wird. In dieser speziellen Muskelverspannung der Aryknorpel sehen wir einen Selbstregulationsmechanismus, welcher die Schwingungen derart dämpft, dass ein Überschlagen der Stimmlippen über die Medianlinie und an die Seitenwände sowie atypische Schwingungsformen verhindert werden.

Über die Bedeutung der endolaryngealen Muskulatur für den Schwingungsvorgang sind seit Ferrein im Jahre 1741 einem Leichenkehlkopf den ersten Ton abgerungen hat und damit erstmalig den Beweis erbracht hat, daß die Stimmbänder auch passiv durch den Luftstrom erregt werden können, verschiedenste und oft im Widerspruch stehende Meinungen vertreten worden. Joh. Müller hat auf Grund seiner Experimente an isolierten Menschenkehlköpfen bereits erkannt, daß eine Tonerhöhung durch Zunahme der Spannung der Stimmbänder gesetzmäßig eintritt, eine solche aber auch durch Erhöhung des subglottischen Druckes möglich ist. Bei letzterem Vorgang erfolgt seiner Meinung nach eine Verengung des subglottischen Lumens. Dieser Erklärung hat Rinne widersprochen. Er meinte, daß bei den Experimenten von Joh. Müller der erhöhte Seitendruck die schwingenden Anteile der Stimmbänder verdünnt und daher die Erhöhung der Anblaseluft eine Erhöhung des Tones nach sich zieht. Damit hat Rinne erstmalig das Prinzip einer Art Dämpfung der Stimmbänder und dadurch eine Verringerung der schwingenden Massen der Stimmbänder in Betracht gezogen. Fernald hat dann an seinen eigens konstruierten Pfeisterpfeifen mathematisch nachgewiesen, daß die Frequenz eines solchen Systems nicht nur vom Elastizitätskoeffizienten, sondern auch von der schwingenden Masse abhängt und daß sie mit ihrer Verkleinerung zunimmt (Hulthés). Nach Hulthés wäre eine solche Verminderung der schwingenden Anteile der Stimmbänder unter der Wirkung der eingesetzten Muskeln denkbar. Nach wie vor wird das von Joh. Müller aufgeworfene Problem der Kompensation der physischen Kräfte im menschlichen Kehlkopf als schwächster Punkt der myoelastischen Theorie viel diskutiert. Pressman & Coleman meinen, daß man zu dieser Kompensation entweder die Spannung oder Elastizität des Stimmbandes verringern muß oder das Stimmband muß sich bezüglich der Dicke und Länge ändern oder

es muß dadurch gedämpft werden daß sich Teile beider Stimmbänder eng aneinander legen wodurch die schwingenden Massen in der horizontalen Ebene verkürzt werden. Somit haben diese Autoren zur Klärung des Schwelltones bei welchem also die Lautstärke durch größeren subglottischen Druck bei gleichbleibender Tonhöhe ansteigen soll die Ansicht von Nadolecny die besagt daß dazu die Kontraktion des M. thyroarytaenoides mit auch im gegenteiligen Sinn wie der subglottische Druck sich ändern muß weiterhineingezogen.

Sowohl Pressman & Klemen und Moore sind der Meinung daß die Tonhöhe nicht nur von der Spannung und dem intratrachealen Druck sondern auch von einer gewissen Dämpfung der Stimmbänder abhängig ist. Letztere hat eine Verringerung der schwingenden Masse zur Folge und kann entweder zu einer Verschmälerung der Stimmlippen im Querschnitt führen oder es kommt wie aus dem Hochgeschwindigkeitsfilm der Bell Telephone Laboratories hervorgeht zu einer Aneinanderlagerung der rückwärtigen Anteile der Glottis so daß also nur ganz vorne ein schwingungsfähiger Spalt offen bleibt. Nach Farnborg Andersen ist im Mittel- und Kopfregeister so eine Dämpfung nicht vorhanden. Dies stünde seiner Meinung nach nicht im Einklang mit der Form der Glottis und der zunehmenden elektrischen Aktivität der bei diesem Vorgang beteiligten Kehlkopfmuskeln. Wohl aber muß beim Übergang von einem niederen zum höheren Register eine Verminderung der schwingenden Masse eintreten, da kein Anstieg der elektrischen Aktivität dabei nachweisbar ist. Dies stimmt mit den Untersuchungen von Fuchsinger überein, der feststellte daß die Stimmbänder in tiefen Lagen mit ihrer ganzen Länge im Kopfregeister nur mit den Rändern schwingen. Nach Farnborg Andersen zeigt der high speed motion film der Bell Telephone Laboratories daß die schwingenden Stimmbänderanteile durch einen solchen Dämpfungsprozess im upper thin Register verkürzt werden. Nach Vallinien haben Lomogramme und röntgen kinematographische Filme den Nachweis erbracht daß wenn die Tonhöhe im Brustregister ansteigt die Muskelmasse der subglottischen Gegend nach seitwärts gepreßt wird. Nach Behringer entspringen vom Conus elasticus einzelne Muskelfasern die auch von Floyd Negus & Neil bestätigt wurden und ihrer Meinung nach wohl in der Lage wären den unteren Teil der Stimmbänder zur Seite zu drücken wodurch die vorher erwähnte Verringerung der schwingenden Massen (dünnen Stimmlippen) erzielt werden kann.

Auf Grund von Experimenten mit künstlichen Kehlköpfen hat Smith seine membrane cushion Theorie der menschlichen Stimme aufgestellt. Diese besagt daß zur Stimmbildung in erster Linie eine isolierte schwingungsfähige Schleimhaut notwendig ist. Der Schwingungsstart erfolgt durch aerodynamische Kräfte. Zur Tonerhöhung ist neben der Spannung vor allem die subglottische Dämpfung durch Kontraktion der von Behringer beschriebenen subglottischen Muskelfasern von Bedeutung. Diese Dämpfung hat eine Versteifung des subglottischen Raumes und somit eine Verringerung der schwingenden Masse zur Folge. Für die Anhänger der neurochronaxischen Theorie

(Husson u. a.) ist die Annahme einer solchen Tonerhöhung durch eine muskuläre Dämpfung zur Verringerung der schwingenden Massen nicht erforderlich, da ja eine Erhöhung der Frequenz einfach durch Vermehrung der cerebral gesteuerten rhythmischen Kontraktionen des *M. vocalis* erzielt wird.

Wenn wir also von dieser umstrittenen neurochronaxischen Theorie absehen, so spielt die Dämpfung der Stimmbänder beim Schwingungsvorgang eine wichtige Rolle. Nach den erwähnten Autoren kann somit eine Dämpfung, das heißt Behinderung oder Hemmung der Schwingungsfähigkeit einzelner Teile der Stimmlippen durch den Luftstrom durch direkten Zug bestimmter Muskelfasern oder durch die Apposition der *Cartilagine arytaenoides* erfolgen und führt zur Verminderung der schwingenden Massen. Die Dämpfung kann entweder subglottisch oder in den lateralen Stimmbandanteilen erfolgen oder es werden Anteile der Stimmbänder längs der Glottis durch gegenseitiges Aneinanderpressen vom Schwingungsakt ausgeschlossen.

Wenn es sich bei diesen verschiedenartigen Eigendämpfungen der Stimmbänder um eine Trägheit der endolaryngealen Muskulatur handelt, so mußten bei Lahmungen auch Störungen der Stimmbanddämpfungen nachzuweisen sein. Ich habe nun aus einem Hochgeschwindigkeitsfilm der in Zusammenarbeit mit Berendes, Luchsinger & Phister angefertigt wurde, die Schwingungen ungelahmter Stimmbänder mit solchen bei einer beidseitigen Paramedianlahmung und bei einer Lahmung in Intermediarstellung kritisch verglichen und nach Ausfallerscheinungen in dieser Frage geprüft. Wenn wir nun von dieser Perspektive die normalen Schwingungsverhältnisse im vorgeführten Zeitdehnerrfilm der Stimme einer Sopranistin mit 650 Hz 38 db und 5000 Bildern pro Sekunde betrachten, so fällt uns in erster Linie die völlig symmetrische und beherrschte nach lateral langsam abgedämpfte Schwingungsform auf. Obwohl ich mit dem Stroboskop trotz jahrelanger Untersuchungen niemals mit Sicherheit eine Schwingung in der *Pars cartilaginea* feststellen konnte, zeigt der Hochgeschwindigkeitsfilm eindeutig, daß die *Processi vocales* zuerst eröffnet werden, von da nach vorne die Glottis bis zur Flaschenform sich erweitert und dann von vorne nach rückwärts sich wieder schließt, bis zuletzt die *Processi vocales* aneinander liegen. In der *Pars cartilaginea* bleibt bei dieser Frequenz und Stimmetechnik ständig ein kleiner Spalt offen (Abb. 1). Die Einwärtsschwingung hört genau in der Medianebene auf, so daß man den Eindruck hat, daß sich die Stimmbänder kaum berühren. Die senkrechte Schwingungskomponente ist sowohl bei normalen als auch bei gelähmten Stimmbändern im Hochgeschwindigkeitsfilm weniger gut festzustellen als bei binokularer Betrachtung mit dem Stroboskop.

Im zweiten Teil des Hochgeschwindigkeitsfilmes werden die Schwingungen bei einer beidseitigen Lahmung in Paramedianlage mit einer Arypexie des linken Stimmbandes mit einer Aufnahme Frequenz von 4915 Bildern pro Sekunde und der Tonfrequenz von 308 Hz gezeigt. Dabei fällt auf, daß die Schwingungen des rechten in Paramedianlage gelähmten Stimmbandes nach median auf das lateralierte Stimmband aufrallen und auf diesem

eine nach lateral ziehende oberflächliche Schwingungswelle auslösen. Der horizontalen Schwingungen mit deutlicher vertikaler Komponente ergreifend das Stimmband in voller Breite und schlagen an die laterale Wand, die schließlich mit dem Fächerband auch zu vibrieren beginnt. Auch die Schleimhautfalte in der Tiefe und der Cartilago arytaenoides schwingt im selben Rhythmus mit und der freigebliebene Spalt bei weitester Glottisöffnung spricht dafür, daß die Fächerfasern des paramedian gelähmten Stimmbandes an der Schwingung teilhaben. Interessant ist, daß bei der Außenschwingung der Precoarctation in die Glottis vorspringt, während die Innenschwingung ziemlich flach verläuft. Die Innenschwingung ist parallel erfolgt (Abb. 2). Weiters fällt in dem nach diesen Untersuchungen zu beurteilenden Stadium der Schwingungsänderung im Vergleich zum Normalzustand eine ausgesprochene, hochgradige Asymmetrie der Bandschwingungen in der ersten Hälfte wohl durch die verschiedene Form und verschiedene Spannung der Stimmbänder infolge der einseitigen Operation erklärt werden können.

Wie es sich aus Abb. 2 ergibt, ist die Asymmetrie verschiedenheit und obwohl ja links durch den operativen Eingriff eine noch eine künstliche Dämpfung der dorsalen Stimmbänder vorhanden ist, so ist doch ein Binnenspringen beim Fächerband erfolgt ist, die Frequenzänderung ist aber nicht so stark ist, erscheint vorerst unklar. Wenn wir aber leuchtend sehen, daß die schwächer gespannte Stimmbänder den Fächerband als höherer Frequenz ausstrahlt, ist es wohl durch welchen der subglottische Druck erhöht wird, die Fächerbande springen werden kann, so ist erklärlich, daß die Außenschwingungen der Innenschwingen nur gleichzeitig im Stadium des höchsten subglottischen Druckes erfolgen kann. Entsprechend der verschiedenen Spannung und Dämpfung werden aber beide Stimmbänder mit ungleicher Faserstärkenweite und Form schwingen. Wir sehen also, daß bei dieser Art der pathologischen Schwingungen das myotatische Moment der vermehrten Spannung und Dämpfung nur für die verschiedenen Schwingungsform das aerodynamische Moment aber für die Frequenz beider Stimmbänder maßgebend ist. Da der subglottische Druck immer das Stimmband mit der stärkeren Spannung zuerst zur Seite drückt, muß auch das stärker gespannte Stimmband entweder im gleichen Moment zur Seite schwingen oder wird wenn die Spannung zu stark ist, überhaupt nicht in Partizipation. In einem solchen Schwingungsstillstand bei Lähmung in Partizipation und starker Spannung haben wir in einem Fall Stroboskopie gesehen. Zur Klärung eines solchen Schwingungsstillstandes hat HUSSON, daß die zentralen Impulse fehlen, dieser Vorgang zwanglos durch aerodynamische Gesetze erklärt werden kann.

Demgegenüber verhalten sich die Stimmbänder bei in Intermediarlage und weit offener Glottis völlig beidseitiger Paramedianlähmung und einseitiger Asymmetrie beider Stimmbänder nicht allzu verschieden. Bei thyreomedeus die durch die Recurrenslähmung bedingt ist, das in Intermediarlage gelähmte Stimmband

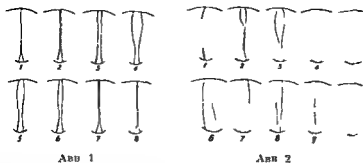


Abb 1

Abb 2

Abb 1 Schwingungen normal beweglicher Stimmbänder bei 650 Hz

Abb 2 Schwingungen von Stimmbändern mit beiderseitiger Lahmung in Form von *aryepexy* und linksseitiger Arypexie bei 308 Hz

außer der Recurrenslähmung auch noch eine Lahmung des *recurrens* Kehlkopfnerven vorhanden sein muß (Hofer & Jeschek) werden in unserem Zeitlupenfilm noch die Schwingungen der *recurrens* bei einer einseitigen Lahmung in Intermediarlage mit hochgradiger *recurrens* bei einer Tonhöhe von 180 Hz und Aufnahme Frequenz von 1000 pro Sekunde gezeigt. Das intermediär gelähmte Stimmband und bewegliche beginnen erst dann zu schwingen wenn die Epiglottis teilweise überdeckt und das rechte nicht gelähmte noch weiter über die Medianlinie adduziert wird. Erst dann ist genügender Überdruck zum Schwingungsstart vorhanden. Da aber der Glottisabschluß erzielt wird ist auch die Frequenz der Schwingungen entsprechend der hohen Spannungsdifferenz verschieden. Die Schwingungen des gelähmten Stimmbandes sind wie aus dem Schema ersichtlich sehr mäßig und können am besten mit einem in Schwingung versetzten Stiel verglichen werden (Abb 3).

Weiters ist im Film noch bemerkenswert daß bevor die Epiglottis über den Kehlkopfengang legt und bevor das rechte ungelähmte Stimmband über die Medianlinie näher an das gelähmte Stimmband verlagert wird bei weit offener Glottis eine rhythmische vom subglottischen Raum bis zum Stimmbandrand oberflächlich ziehende Hohlraumwelle im ungelähmten Stimmband auftritt. Diese vertikale wellenartige Bewegung wurde erstmals von Farnsworth im Bell Telephone Film beschrieben (glottis) und von Smith genauer präzisiert. Das Kuriosum an dem ist daß wir hier nur eine der drei von Smith beschriebenen Schwingungen nämlich die Hohlraumwelle vorfinden während die tiefenkrechte Schwingungsrichtung der Stimmbänder fehlen.

Als auslösende Ursache dieser aufsteigenden Wellenbewegung wohl mit Sicherheit der Luftstrom feststellen da man erkennt zeitig auch das gelähmte Stimmband von diesem zur Seite weg ohne aber in Schwingungen zu geraten. Eine zentral gesteuerte Kontraktionswelle der in Frage kommenden Muskelanteile

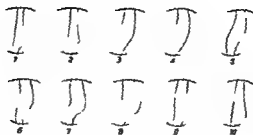


Abb. 3. Schwingungen von Stimmbändern: Stimmkesseltiger Lähmung in Intermedarstellung bei 180 Hz

schließen, daß das Aufsteigen dieser Wellenbewegung bis an den Glottisrand ohne Mitbeteiligung des ganzen *M. vocalis* auch nach der neurochronaxischen Theorie schwer zu erklären wäre. Da in diesem Moment der Filmaufnahme von der Patientin kräftig phoniert wurde, mußte eigentlich nach der neurochronaxischen Theorie eine das ganze normal bewegliche Stimmband ergreifende Schwingung auftreten. Wir sind der Meinung, daß dies deshalb nicht der Fall ist, weil in Folge der weit offenen Glottis ein zu geringer subglottischer Druck vorhanden ist, um das Stimmband in Schwingungen zu versetzen. Wohl aber ist der intratracheale Luftstrom in der Lage, den elastischen Vorsprung der Stimmlippen rhythmisch einzudrücken und vorübergehend den Stimmbandrand wie Ringe angenommen hat zu verschmälern. Diese Erscheinung läßt sich also nach der aerodynamisch-myoeelastischen Theorie zwanglos erklären.

Wir wollen im Rahmen unserer Untersuchungen vor allem darauf hinweisen, daß bei Lähmung der inneren Kehlkopfmuskulatur in erster Linie die Schwingungsdämpfung nach median und lateral ausfällt, so daß wir also mit größter Wahrscheinlichkeit sagen können, daß diese Dämpfung muskular bedingt sein muß. Natürlich dürfen wir auch bei dieser Frage die Mitbeteiligung aerodynamischer Einwirkungen durch die Abänderung der Glottis und der subglottischen Räume nicht ganz außer acht lassen. Wir glauben aber, daß für diese bei steigender Tonhöhe stärker wirkende Dämpfung der lateralen Stimmlippenanteile in erster Linie die von mir bereits beschriebene antagonistische Zugelwirkung vom *M. thyroarytenoideus* int. und *externus* maßgeblich ist (Abb. 4). Auf Grund von Versuchen an Kehlkopfspreparaten, wobei die Muskelnstücke mit Haltefäden befestigt wurden, konnte von mir festgestellt werden, daß bei Zug an den Internusfasern ein Gegenzug an den Lateralfasern spürbar wurde, somit also diese beiden Muskeln außer ihrer gemeinsamen adduktorischen Wirkung auch eine antagonistische Komponente besitzen. Auf die Bedeutung dieser Gegenwirkung für den fließenden Ablauf der Adduktion und Abduktion der Stimmbänder, so wie zur Fixation der *Cartilagoes arytenoidei*, habe ich bereits hingewiesen.

Aber auch der Schwingungsablauf der Stimmbänder wird durch diese antagonistische Funktion dieser beiden Muskeln und der verschiedenen ansetzenden Muskelanteile des *M. vocalis* bestimmt. Während die medialen Anteile



Abb. 4

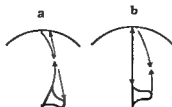


Abb. 5

Abb. 4 Zügelwirkung von *M. thyroarytaenoideus internus* und *externus*

Abb. 5 Antagonistische Zügelwirkung der medialen und lateralen Muskelfasern in der Stimmrinne a) bei Auswärtsschwingung b) bei Einwärtsschwingung

des *M. vocalis* auch ohne der von Goertler angenommenen und umstrittenen Muskelfaseranordnung eine stärkere Abduktionskomponente haben werden die lateralen Anteile ähnlich wie die Lateralfasern rein adduktorisch wirken. Beim Auswärtsschwingen der medianen Fasern des *M. vocalis* werden die lateralen *Vocalis*- und Lateralfasern gedehnt und dadurch je weiter die Schwingung nach auswärts schreitet umso stärker gedämpft, auch wenn die Spannung beider Muskelanteile gleich bleibt. Haben die Schwingungen auf die lateralen Muskelfasern übergreifen, werden die medialen Fasern gedehnt und durch die Spannungszunahme ebenfalls gedämpft (Abb. 5). Durch diesen sinnvollen Schwingungsmechanismus wird sowohl das Anschlagen der Schwingungen an die seitliche Kehlkopf wand als auch das Überspringen der Schwingungen über die Medianlinie verhindert. Weiters aber werden bei zunehmender Spannung der äußeren Fasern die lateralen Anteile der Stimmlippen immer mehr gedämpft, bis schließlich in den höchsten Lagen nur mehr die von Buchsinner nachgewiesenen Randschwingungen übrigbleiben.

Die von mir beschriebene zum Teil auch antagonistische Wirkung aller Adduktoren wird ebenso in der Lage sein störende Eigenvibrationen durch Muskelkontraktionen zu dämpfen, da jedem Muskelzug ein Gegenzug entspricht.

Zum Schluß möchten wir nochmals die von den erwähnten Autoren angegebene Dämpfungsmöglichkeit der Stimmbänder und unsere Beobachtungen über diesen Vorgang kurz zusammenfassen: 1. eine Dämpfung der Stimmlippen kann erfolgen: 1. Durch Erhöhung des intratrachealen Druckes (Rinne, subglottische Hohlraumwelle nach Farnsworth und Smith); 2. durch bestimmte in den Conus elasticus einstrahlende Muskelfasern, wodurch die Stimmbänder verdünnt und gedämpft werden (Pressman & Coleman, Lloyd, Negus & Neil und Behringer); 3. durch die oben beschriebene antagonistische Wirkung von *M. thyroarytaenoideus internus* und *externus*, wobei die lateralen Stimmlippenanteile und Eigenvibrationen der Stimmbänder gedämpft werden; 4. durch die erhöhte äußere und innere Spannung, oder beides zusammen; 5. durch das Aneinanderpressen korrespondierender Anteile der Stimmlippen.

bänder. Dabei kommt es zur Fixation der Glottis cartilaginea oder es werden noch zuzuglich Teile der ligamentösen Stimmbänder immobilisiert

Dieser Dämpfungsmechanismus hat sowohl die Aufgabe für eine störungsreiche Schwingungsmöglichkeit der Stimmlippen zu sorgen, als auch durch Verringerung der schwingenden Massen und bei gleich bleibender Tonhöhe — aber Abänderung der Lautstärke — die von Joh. Müller geforderte „Kompensation der physischen Kräfte im menschlichen Kehlkopf“ zu gewährleisten

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DISKUSSION

G. Kelemen Der Orang besitzt einen sehr effektvollen Dämpfungapparat indem die kaudale Schaufel der Epiglottis mit seinen Blättern die Plicae ventriculares ausfüllt

Die „consensualen“ Kurven von Nadoleczny (aufgenommen vor den Larynx des stummen Zuhörers) sind im Zusammenhang mit der Theorie von Husson zu bewerten

A. Sercer Zu dem wunderbaren Film vom Kollegen Jeschek muss man ihm gratulieren. Man bedenke dass als Grundlage dieses Films fünf Tausend Aufnahmen pro Sekunde dienen. Das ist sicher ein Prachtstück der kinematographischen Technik. Aber nach wie vor kann ich mich noch nicht weiter für die eine noch für die andere Theorie über die Entstehung der Stimmenbandschwingungen entscheiden, trotzdem uns der gesehene Film viel zum Nachdenken gibt. Wir können leider noch zu wenig Methoden welche uns ermöglichen wurden eine ungezwungene Entscheidung zu treffen

Am Anfang des Films haben wir Stimmbandsschwingungen in einem normalen Kehlkopf gesehen. In diesem Falle war der subglottische Druck bestimmt normal aber in beiden pathologischen Fällen war der subglottische Druck bestimmt sehr niedrig. Trotzdem konnte ich am Film keinen wesentlichen Unterschied in der Amplitude des gesunden Stimmbandes bemerken. Deswegen erlaube ich mir die Frage wie hoch, oder besser gesagt, wie tief war der subglottische Druck in beiden pathologischen Fällen?

M. Arslan I congratulate Dr. Jeschek on his very interesting speech and film. May I ask Dr. Jeschek if he could measure, in the photograms of the high speed picture illustrating the vibration pattern of the vocal cord by recurrent one sided surgical interruption, the vibration frequency of the mucous border of the paralyzed and of the sound cord. In fact, in cases with functional compensation of the paralyzed cord, permitting a voice formation, if the frequency of the two cords is even approximately the same, this finding becomes a fundamental disproof of Husson's theory of *coup par coup*. The reason for introducing the Jeschek film would be the demonstration that a vocal function can be restored even if there are no nervous impulses to the laryngeal muscles.

J. Jeschek (Antwort) Herrn Prof. Kelemen danke ich für seinen interessanten Hinweis auf den Bau des Kehlkopfes bei Anthropoiden.

Ebenso danke ich allen Herren, die sich an der Diskussion beteiligt haben. Ich bin überzeugt, daß nur in Zusammenarbeit mit Allen, die an diesem Problem interessiert sind, weitere Fortschritte in der Forschung erreicht werden können. Vorwegnehmen möchte ich gleich, daß ich mit meinen Ausführungen nicht die Absicht gehabt habe, die neuro-chronaxische Schwingungstheorie zu widerlegen, sondern nur aufzeigen wollte, daß alle von mir an gelähmten und ungelähmten Stimmbändern gemachten Beobachtungen durch eine Kombination der aerodynamischen mit der myoelektischen Theorie ohne Zwang zu erklären waren. Es ist daher kein Grund vorhanden, neue theoretischen Prinzipien heranzuziehen. Doch müssen wir bei der Vielfalt jedes biologischen Vorganges daran denken, daß infolge mehrerer in einem Organ vereinten Funktionen und im Interesse der Sicherung einer Organfunktion für einen Vorgang aetiologisch verschiedene Ursachen vorhanden sein können. Weiters sollten wir auch bei einer negativen Beurteilung der neuro-chronaxischen Theorie nicht von vornherein alles ablehnen, sondern man mußte auch noch die Möglichkeit ins Auge fassen, daß die Stimmbänder nur in einem kleinen Frequenzbereich, welcher den Aktionsströmen entspricht, schwingen und die weiteren Frequenzen ähnlich wie bei der singenden Säge, lediglich durch Spannungsdifferenzen erzielt werden. Ich habe mich mit dieser Frage an Van den Berg, den überzeugtesten Gegner der neuro-chronaxischen Theorie gewandt, mit der Bitte auch in dieser Richtung seine Untersuchungen anzustellen, da ich der Meinung bin, daß diese Frage in erster Linie von Physiologen geklärt werden mußte.

Damit habe ich Prof. Sercer auf eine seiner Fragen bereits geantwortet. Bezüglich des subglottischen Druckes war Prof. Hennebert so freundlich, bereits eine Antwort zu geben. Über das Maß des subglottischen Druckes bei Phonation wurden von mehreren Autoren bei tracheotomierten Patienten nähere Angaben gemacht. Bei unseren drei im Hochgeschwindigkeitsfilm gezeigten Fällen ist es wohl ganz klar, daß dort wo die Glottis geschlossen werden kann, wie bei den ersten beiden Fällen, der sub

glottische Druck größer sein muß, als im letzten Fall wo durch die Lähmung die Glottis weit offen stand

Zur Bemerkung von Herrn Prof. Arslan möchte ich noch hinzufügen, daß die synchrone Gegenschlagsbewegung gleicher Frequenz, bei verschiedener Spannung beider gelähmten Stimmbänder oder bei einseitiger Lähmung, in erster Linie vom kompletten Glottisschluß abhängig ist, somit also auch dieses Phänomen ganz im Sinne von Herrn Prof. Arslan durch aerodynamische Vorgänge zu erklären ist

RI FLEXIONS SUR LE « STRESS »

G CAMBRILLIN
Bruxelles Belgique

L'auteur définit ce qu'il entend exactement par « stress » et ses conséquences, de même qu'il décrit les moyens physiologiques de combattre le « stress » ceux-ci sont en somme « les phénomènes d'adaptation » (Seize)

Il décrit le mécanisme du « stress » dans lequel interviennent en ordre principal certaines hormones (voie humorale) et le sympathique (voie nerveuse)

Il parle également des « facteurs de conditionnement » agents modificateurs des phénomènes d'adaptation

A mon avis on a eu tort de traduire le mot « stress » par agression ou attaque il faudrait plutôt dire « alerte alarme choc » ou simplement « tension » qui est une des traductions de l'anglais

Le « stress » est la conséquence sur l'organisme d'une agression ou attaque par des agents divers (émotion intervention chirurgicale coups froid chaud microbes). C'est en somme un état d'alerte d'alarme de l'organisme qui lui fait prendre des dispositions adéquates pour y pallier. Et ces dispositions sont les « phénomènes ou réactions d'adaptation » qui représentent la mobilisation de tous les moyens de défense dont dispose l'organisme

Tout agent qui attaque l'organisme a deux effets

1° Un effet spécifique

2° Une réaction non spécifique qui se manifeste par un syndrome général d'adaptation

Le stress se manifeste en somme de triple façon : a) par une action directe sur l'organe intéressé ; b) par une action par voie humorale (hormones) ; c) par une action par voie nerveuse (sympathique) (système neuro végétatif)

La pathogenèse du « stress » est tripartite

1° Un effet direct de l'agent externe

2° Inhibition de cet effet par des agents endogènes c'est l'immunité ou protection

3° Action des facteurs endogènes favorisants : sensibilisation ou allergie

Les voies humorales de production des agents « stressants » passent par l'hypophyse antérieure et la cortico surrénale au niveau desquelles sont élaborées la majorité des hormones d'adaptation et parmi celles-ci l'ACTH (hormone adréno-corticotrope) et les corticoides anti-phlogistiques (cortisone) qui inhibent les phénomènes inflammatoires tandis que l'hormone somatotrope (HST) et les corticoides pro-phlogistiques (desoxycorticostérone) les stimulent

Ces hormones sont de ce fait appelées hormones d'adaptation car elles complètent ou assument seules les réactions d'adaptation physiologiques de



Fig 1



Fig 2



Fig 3

plissage et les pulsations augmentaient pendant la compression (fig 3) La malade ressentait alors une tension désagréable dans la tête

Le radiogramme de l'os temporal gauche (fig 4) démontrait dans l'écaille de l'os temporal au dessus de l'angle sinodural plusieurs raies transparentes en forme d'étoile évidemment les sillons et canaux des veines multiples et dilatées

Au repos la malade ne se rendait compte d'aucun bourdonnement d'oreille mais à l'attention concentrée elle percevait un profond bruit rythmique qu'elle comparait au ronron d'un chat À l'effort elle ressentait en avant du pavillon gauche les pulsations synchroniques avec le pouls

Le phonendoscope rendait bien audible le bruit objectif dans tout le voisinage du pavillon avec un maximum en avant du tragus

La phonangiographie avec l'appareil multiscriptor décèle un bruit dans toutes les fréquences avec enregistrement optimal dans les fréquences plus élevées (fig 5) Il s'agissait du bruit continu systolodiastolique qui est caractéristique pour la fistule artério-veineuse (Dr Kreilek)

À l'aide d'oxymétrie par méthode polarographique on a établi la saturation du sang par l'oxygène dans la veine du bras saturation de 28%, dans la veine jugulaire int de 98%, et dans l'artère fémorale de 100%.

La saturation du sang par l'oxygène constatée dans la veine jugulaire interne témoigne de l'existence d'une fistule artério-veineuse considérable dans la région de la veine jugulaire interne

La température de la peau à la tête et au cou était la suivante (Dr Kreilek)

Tête et cou	A droite (°C)	A gauche (°C)
La joue	29.5	32
Pavillon	31.0	34
Cou	32.0	33.5

Menton 32°C

Examen cardiologique cœur non hypertrophié ECG normal Pouls 88/min Tension sanguine 160/70

Examen neurologique (clinique neurologique) Point de symptômes intracraniaux (cerbreaux) pathologiques Plus tard on a examiné aussi le fond de l'œil (voire la constatation après la seconde opération)



fig. 4

l'angiographie (service radiologique UN, Dr Rosch) On a injecté dans l'artère carotide commune 10 ml de Diodone Spofa à 40%. La matière contrastante est injectée tour à tour dans les deux carotides communes.

A l'*arteriographie droite* (fig. 6) dans l'incidence anteropostérieure on aperçoit le remplissage contrastant dans toutes les branches de la carotide externe dont le diamètre est normal. De sa branche parieto occipitale la matière contrastante passe dans la région parieto occipitale gauche ou au dessus du conduit auditif externe elle alimente des vaisseaux qui sont dilates de façon cavernueuse dans une étendue de 6×4 cm. Les vaisseaux afférents ont un diamètre de 1 à 2 mm.

La veine efférente dont le diamètre est de 6 mm prend la direction caudale et retrorotulaire. Les veines cérébrales et les sinus visibles sans modifications.

Le siphon de la carotide interne droite est sans modifications de même que l'artère cérébrale antérieure et moyenne. L'artère cérébrale antérieure gauche se est remplie en partie par la intermédiaire de la communicante antérieure.

La matière contrastante injectée dans la carotide commune gauche (fig. 7) ne

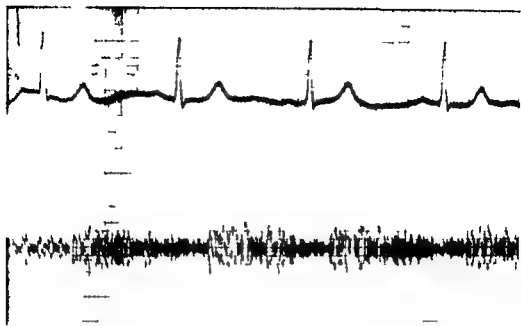


FIG 5

remplissant bien que sa branche externe (fig. 7a). La carotide interne (fig. 7b) n'est remplie que faiblement et son diamètre est plus petit parce que la plus grande quantité du sang est divertie par la carotide externe dont le diamètre atteint 7 mm.

La matière contrastante remplit les vaisseaux à dilatation cavernueuse dans une grande étendue (fig. 8a). Elle remplit la malformation vasculaire qui déjà pendant l'arteriographie droite se remplissait par deux branches arrivant de la région parieto occipitale droite. Dans la troisième seconde la matière contrastante passe dans les veines efférentes dilatées variqueuses d'un calibre jusqu'à 8 mm. Leur trajet est irrégulièrement sinueux (fig. 8b). La matière contrastante est conduite dans la veine jugulaire superficielle gauche qui atteint un calibre de 13 mm (fig. 8c).

En résumé on peut dire l'arteriographie bilatérale exécutée pour hémangiome cavernueux de grandes dimensions de l'oreillet de son entourage a établi son alimentation par la carotide externe gauche et dans la région parieto occipitale par deux branches cutanées plus petites arrivant de la région parieto occipitale droite. En même temps on a trouvé une disproportion considérable entre la quantité sanguine de la carotide interne et externe gauche.

On a fait jusqu'à présent deux opérations.

Operation I (13.4.60) Par une incision près du bord antérieur du muscle sterno-cléido-mastoïdien à gauche on a d'abord exposé la veine jugulaire externe antérieure et la veine jugulaire interne considérablement dilatée. En pénétrant jusqu'à la bifurcation des carotides on a constaté la carotide externe était



Fig 6

Fig 7

venifiee immediatement au dessus de la bifurcation et il diffusent dans une longueur d'environ 2 cm et sa paroi etait moins resistente. Entre la carotide externe (fig 9a) et la veine jugulaire externe anterieure (fig 9b) il y avait plusieurs courtes et directes communications fistulaires. Les memes communications fistulaires courtes et directes (1 a 3 mm) existaient entre la carotide externe et la v. jugulaire interne (fig 9c.) Elles



Fig 8



Fig. 9



Fig. 11

Avant l'un des artères leur paroi était plus épaisse que celle des veines (artérialisation). Sur la v. jugulaire interne exposée on observait distinctement le froissement crû par les tourbillons du sang pénétrant par les communications fistulaires dans la v. jugulaire interne. L'artère carotide externe est ligaturée au dessus du détachement de l'artère linguale. Les communications fistulaires artério-veineuses ont été sectionnées et autant que possible ligaturées. Il fallait fermer les très courtes fistules après leur section par une suture de la paroi des vaisseaux respectifs. L'hermeture complète de la plaie. Après l'intervention le pavillon était plus petit et plus pile sa surface moins tendue.

Operation II (25 60) Par une incision derrière le sillon rétroauriculaire et parallèle avec lui à gauche au dessus du paquet des veines dilatées on a réséqué la tumeur vasculaire au dessus de la mastoïde et à la face postérieure du pavillon avec interruption progressive des vaisseaux marginaux leur ligature ou suture. On a laissé en place les communications veineuses entre les vaisseaux dans l'oreille de l'os temporal et la veine jugulaire externe latérotorotale. L'hémostase de l'hémorragie provenant des varices rompues fut effectuée par la ligature perivascularaire. Suture de la plaie drainage par un drain en verre.

Examen histologique (Prof. agrégé Valach) Il a plupart des parois des cavités vasculaires a le caractère des veines (fig. 10) sans doute d'un anévrysme artério-veineux avec modifications sclérotiques avancées dans le segment veineux. Dans les parois de la formation tumorale on a constaté une lamelle élastique (signe d'artérialisation des parois veineuses).

I^{er} cours postopératoire après la première opération (13-4) fut sans complications la plaie guérit per primam. Le lendemain de l'opération il y avait dans



FIG 10

l'oreille gauche un bruit fin, différent de celui d'avant l'intervention. Après cette première opération le profond bruit systolique était audible objectivement au niveau de l'autre.

Après la seconde opération (2/5) la plaie se cicatrisait sans réaction inflammatoire. Sur la suture apparurent des petites nécroses circonscrites, mais la plaie ne s'ouvrit pas.

Le 20 mai (donc 18 jours après la seconde opération) il se développa à l'endroit d'une varice dans le sillon rétroauriculaire une nécrose circonscrite (fig. 11) de la peau avec hémorragie, qu'on arrêta par une légère compression. Une autre nécrose limitée de la paroi variqueuse se développa plus tard sur le bord de l'helix. Les plaies provenant de ces nécroses ne se cicatrisaient que très lentement.

Malgré cette circulation évidemment affaiblie du pavillon, les petites corrections locales (réduction du lobule, quelques petites varices) ont été remises à plus tard.

Dans la période suivant la seconde opération on a contrôlé la circulation intracrânienne par l'examen du fond de l'œil pendant la compression des veines efférentes de l'endocranium, c'est-à-dire les deux veines jugulaires internes et dans ce cas aussi la veine jugulaire externe dorsolatérale gauche.

Sur le fond de l'œil à gauche on a constaté les veines quelque peu dilatées qui après compression de la v. jugulaire externe, se remplissaient plus abondamment tandis que les ondes pulsatoires s'aplatissaient et finissaient par disparaître.

Pendant la compression de la veine jugulaire interne gauche on observait

un changement transitoire de la pulsation dans la veine carotidienne gauche pulsation disparaissant pour la durée de trois à quatre battements puis reparaissait une onde pulsatoire d'abord plate dans la suite de la normale.

Sur le fond de l'œil à droite la compression de la veine jugulaire externe gauche ni celle des veines jugulaires internes de deux côtés n'entraînent aucun changement essentiel du remplissage veineux.

Analyse

D'après les résultats des investigations cliniques et de laboratoire il s'agit d'une angiome artérioveneux de grandes dimensions. Bien que l'anamnèse ait mentionné l'érysipèle comme sa cause originelle il n'y a pas de doute vu les constatations opératoires et histologiques qu'il faut supposer un trouble de développement avec formation des fistules artérioveneuses et d'une tumeur caverneuse et racineuse.

Ces tumeurs sont rares. Olivecrona en a trouvées parmi 1000 cas de tumeurs cérébrales 125 dont sept dans la région de la carotide externe. Moi-même j'ai observé au cours de 41 ans trois cavernomes artérioveneux localisés à l'oreille externe. Dans tous les trois cas on a pu constater une autre affection: une fois c'était la carotide externe une autre fois l'artère temporale superficielle la troisième fois l'artère auriculaire postérieure. Dans les deux derniers cas il n'y avait que des symptômes locaux et le traitement chirurgical (extirpation) était facile.

Des modifications hémodynamiques essentielles ne se rencontrent que dans les cas des tumeurs plus étendues comme celle qui fait l'objet du présent rapport. Il est évident que cette tumeur se développait d'abord de manière latente avant de pénétrer dans la peau. Les courtes communications directes et les formations de la tumeur vasculaire ont en principe la structure des anastomoses artérioveneuses qui peuvent être elles aussi de forme droite ou sinueuse (outre les formations en glomus). Reste à savoir s'il y a quelques rapports de développement entre les formations normales et pathologiques.

Ce qui est remarquable dans notre cas c'est l'effet hémodynamique produit par les modifications de la région de la carotide externe sur celle de la carotide interne. C'est sans aucun doute l'influence des altérations pathologiques de la carotide externe localisées immédiatement au dessus de la bifurcation de deux artères carotides. On sait aussi que l'interruption du passage dans la carotide externe par une ligature dans ce segment peut avoir une influence défavorable sur la circulation dans la carotide interne.

En outre à la diversion considérable du sang par l'intermédiaire de la carotide externe participaient aussi les veines efférentes très dilatées. S'il n'y avait pas dans notre cas de graves troubles fonctionnels dans le domaine de l'artère carotide interne on peut l'expliquer par le fait que les modifications pathologiques se développaient dès la première enfance lentement et progressivement de sorte qu'elles ont pu être suffisamment compensées. Cette compensation avait tout de même certaines limites. Surtout après la compres-

sion de la veine jugulaire externe gauche on observait des modifications venostatiques sur le fond de l'œil gauche. La malade se plaignait alors d'une sensation désagréable dans la tête. Ce phénomène suggère l'idée qu'on peut par ce procédé et en observant le fond de l'œil après compression de la veine jugulaire interne du côté sain constater l'occlusion du sinus sigmoidal et de la veine jugulaire (par ex par une thrombose) de façon análogue comme dans l'expérience de Queckenstedt.

Les anévrysmes artério-veineux entraînent une augmentation de l'activité cardiaque. Par conséquent il y a souvent des signes d'hypertrophie du cœur avec compensation ou décompensation. Dans notre cas il n'y avait pas de signes du surmenage cardiaque sauf une légère tachycardie. On n'a constaté ni hypertrophie, ni décompensation.

Les anévrysmes artério-veineux s'accompagnent souvent d'un autre symptôme grave qui est l'hémorragie. Dans la région de la carotide externe celle-ci se produit lorsque la tumeur a pénétré jusqu'à la peau dans laquelle se développent des varices à parois très minces et fragiles qui se rompent au moindre traumatisme presque spontanément et entraînent une hémorragie pouvant menacer sérieusement la vie. Cette hémorragie est la cause la plus fréquente de l'intervention chirurgicale d'urgence. Il en était ainsi dans notre cas.

Un autre symptôme important de cette affection localisée à la tête est le *bourdonnement d'oreille*. Comme symptôme objectif il est constant dans toutes les tumeurs accessibles à l'auscultation, mais à la tête il se manifeste souvent aussi un bruit subjectif qui peut gêner considérablement le malade (Oliverson). Dans notre cas son intensité était minime. Il n'y a pas de doute que plus la tumeur est proche de l'oreille interne, plus le bruit vasculaire subjectif est fort. Dans les modifications localisées à l'oreille interne on ne constate que les bruits subjectifs.

Le bruit objectif dans les anévrysmes artério-veineux est si caractéristique qu'il constitue un signe diagnostique important pour établir la nature de l'affection fondamentale en différenciant le bruit vasculaire du bruit cardiaque.

Notre cas démontre aussi les problèmes thérapeutiques que nous posent ces affections qui à la tête ont un but cosmétique, outre la suppression des hémorragies des troubles fonctionnels, surtout intracrâniens et des bourdonnements d'oreille. C'est en première ligne l'élimination de l'artère affectée et la résection de la tumeur. Parfois il est nécessaire de couvrir le défaut cutané provenant de la résection de la tumeur par une plastique. Les nécroses par modifications trophiques constituent un problème thérapeutique spécial.

Dans le cas décrit on a fait usage de deux premières méthodes mentionnées de la ligature et de l'extirpation. Il faut aussi prendre en considération la trophique affectée du segment atteint. Pour les tumeurs de petites dimensions le problème thérapeutique est relativement simple. Dans les affections plus étendues il faut procéder très prudemment, par étapes et avec des pauses.

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RECHERCHES AUTORADIOGRAPHIQUES SUR LA FIXATION DU P^{32} PAR LES CARCINOMES LARYNGIENS ET LEURS GANGLIONS LYMPHATIQUES SATELLITES

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L'étude comparative autohistoradiographique et histologique des carcinomes laryngiens et des lymphonodes régionaux de patients auxquels il avait été administré du P^{32} par voie orale avant l'intervention a démontré que la fixation du radioisotope est particulièrement élevée dans les nœuds néoplastiques du larynx et des lymphonodes satellites.

Ces résultats justifient des ultérieures épreuves dans le but d'une éventuelle utilisation clinique de la méthode des isotopes radioactifs comme moyen complémentaire et collatéral pour le diagnostic des tumeurs malignes du larynx et de leurs métastases.

La détermination de la nature et du siège des tumeurs malignes peut être accomplie aujourd'hui dans beaucoup de cas au moyen de substances radioactives qui prennent part aux procès de croissance cellulaire et utilisent ainsi l'activité exaltée proliférative et métabolique des cellules néoplastiques (Scopinaro et coll.).

Le P^{32} est particulièrement employé dans ces recherches. Ce radioisotope est caractérisé par une demi-période de 14 jours, émet seulement de radiations β peut être donné aux patients avec un bon marge de sécurité, se répand facilement dans les liquides organiques et dans les tissus où il prend part à beaucoup de procès de phosphorylation cellulaire. Il est rapidement absorbé par les os, les organes hématopoïétiques et les tissus en prolifération. Il est employé pour le diagnostic des tumeurs parce qu'il est fixé électivement par les cellules néoplastiques grâce aux plus actifs procès de phosphorylation se développant dans les éléments cellulaires de la tumeur.

Avec les nombreux travaux concernant le diagnostic des tumeurs par les radioisotopes en général et par le P^{32} en particulier dans les autres régions de l'organisme, fait un remarquable contraste l'absence de pareilles contributions en otorhinolaryngologie. Il y a seulement à notre connaissance les expériences de Klein et coll. qui utilisaient le P^{32} pour le diagnostic des métastases carcinomatiques de la tête et du cou et qui constamment observèrent une radioactivité augmentée dans le siège des métastases.

C'est pour cela que nous nous sommes proposés de nous occuper du problème du diagnostic des tumeurs malignes du larynx et du pharynx par les isotopes radioactifs.

Dans des précédentes recherches nous avons étudié, après administration

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RECHERCHES AUTORADIOGRAPHIQUES SUR LA FIXATION DU ^{131}I PAR LES CARCINOMES LARYNGES ET LEURS GANGLIONS LYMPHATIQUES SATELLITES

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L'étude comparatif autohistoradiographique et histologique des carcinomes laryngiens et des lymphonodes régionaux de patients auxquels il avait été administré du ^{131}I par voie orale avant l'intervention a démontré que la fixation du radioisotope est particulièrement élevée dans les plastiques du larynx et des lymphonodes satellites.

Ces résultats justifient des ultérieures épreuves dans le but d'une telle utilisation clinique de la méthode des isotopes radioactifs comme moyen complémentaire et collatéral pour le diagnostic des tumeurs malignes du larynx et de leurs métastases.

La détermination de la nature et du siège des tumeurs malignes est accomplie aujourd'hui dans beaucoup de cas au moyen de substances actives qui prennent part aux procès de croissance cellulaire et utilisent l'activité métabolique et proliférative des cellules (épithéliales, plasmiques, fibrocytes et coll.).

Le ^{131}I est particulièrement employé dans ces recherches car il est caractérisé par une demi-vie de 14 jours et met seulement des fractions de seconde pour être donné aux patients avec un bon marge de sécurité. Il se fixe facilement dans les liquides organiques et dans les tissus où il intervient dans beaucoup de procès de phosphorylation cellulaire. Il est rapidement absorbé par les os, les organes hématopoïétiques et les tissus en pleine croissance. Il est employé pour le diagnostic des tumeurs parce qu'il est fixé

radioisotopes en général et par le ^{131}I en particulier dans les tumeurs malignes de l'organisme. On a remarqué un contraste frappant entre la présence de l'isotope dans les tumeurs en otorhinolaryngologie. Il y a seulement une notre connaissance des expériences de Kiehn et coll. qui utilisaient le ^{131}I pour le diagnostic des métastases carcinomateuses de la tête et du cou et qui constataient une radioactivité augmentée dans le siège des métastases.

C'est pour cela que nous nous sommes proposés de nous occuper de l'étude du diagnostic des tumeurs malignes du larynx et du pharynx au moyen des isotopes radioactifs.

Dans des précédentes recherches nous avons étudié l'absorption et la

préopératoire de P^{32} à des patients atteints par un carcinome du larynx la radio activité du tissu néoplasique extirpé et homogénéisé. Les mesurages révélèrent que les régions laryngées atteintes par le processus néoplasique fixent le radio isotope d'une mesure constamment et significativement plus élevée que les régions normales limitrophes. Cette activité de fixation du P^{32} était moins accentuée lorsque la tumeur avait été précédemment soumise à une intense roentgentherapie préopératoire (Pallestrini, Cremonesi et coll.)

Nous rapportons ici les résultats de recherches autoradiographiques effectuées sur des fragments de carcinomes du larynx et de ganglions lymphatiques satellites. En considérant les buts de nos recherches exécutées surtout pour établir la possibilité d'un éventuel emploi clinique de la méthode des radio isotopes comme *moyen complémentaire et subsidiaire* pour le diagnostic des tumeurs du larynx nous nous sommes bornés à préciser et objectiver la présence et la répartition de la substance radioactive dans le contexte des tissus sans étendre l'investigation jusqu'au niveau cytologique c'est à dire sans étudier la fine distribution du radioisotope dans les diverses parties de chaque élément cellulaire.

À 20 patients porteurs d'épithéiomas laryngés localisés à des sièges divers le P^{32} (500-1000 microcuries selon le poids) fut administré 24 heures avant l'intervention par voie orale ou intraveineuse sous forme d'orthophosphat « carrier free ». Chez 12 patients on pouvait palper des ganglions latrocervicaux agrandis qui chez six patients présentaient des caractères cliniques de ganglions métastatiques. Chez les autres huit patients la présence de ganglions lymphatiques agrandis fut observée en cours d'intervention. Des sections histologiques sériees furent exécutées soit de la tumeur laryngée soit des ganglions extirpés ces sections furent utilisées alternativement pour la coloration usuelle (hématoxyline eosine) et pour la préparation de l'autoradiographie par la méthode ainsi dite « stripping film technique ». Par cette méthode la présence du P^{32} est révélée par un noircissement plus ou moins intense et irrégulièrement distribué selon la variable fixation de la substance radioactive par les éléments cellulaires. Il était ainsi possible de localiser exactement dans le contexte des tissus examinés le P^{32} en comparant l'autoradiographie avec le préparat histologique coloré.

Resultats

Tissu laryngé

Les résultats obtenus se sont répétés avec uniformité dans tous les cas examinés. Dans les autoradiographies il nous a été possible d'observer un noircissement irrégulièrement distribué avec des concentrations massives dans quelques champs et moins marquées dans des autres, ou le noircissement était moindre ou même manquait tout à fait.

La comparaison soigneuse avec le préparat histologique coloré révélait que ce noircissement était évident au plus haut degré au niveau des aires où était localisée le tissu néoplasique tandis qu'il était moins accentué ou même absent dans les aires de tissu connectif internéoplasique.

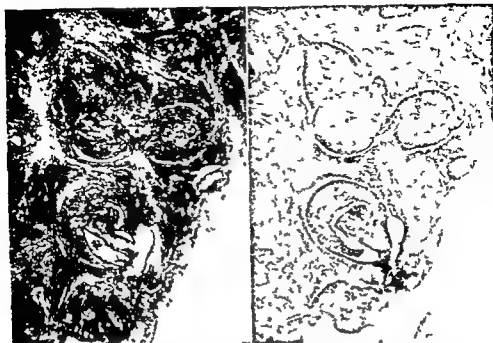


Fig. 1. Détail d'un carcinome du larynx. À gauche autoradiographie à droite coloration à l'hématoxyline. L'accumulation du P³² est particulièrement évidente dans les nodules néoplastiques. L'III est plus accentuée dans les parties périphériques solides des nodules et moins accentuée dans les parties centrales où il y a des pertes épithéliales.

On doit souligner qu'une certaine radioactivité est manifestée par les aires de tissu de type inflammatoire qui peuvent entourer les îlots de tissu néoplasique mais cette radioactivité tout au moins l'examen autoradiographique est incomparablement moins accentuée que celle produite par le tissu tumoral.

En outre la radioactivité est moins marquée dans les foyers de nécrose et fusion néoplasique dans les agglomérats de cellules épithélio-mateuses hyperkératinisantes ou lorsqu'on avait fait auparavant de la roentgentherapie (fig. 1 et 2).

Ganglions lymphatiques

Dans les ganglions lymphatiques satellites même s'ils ne sont pas siège de métastase on observe constamment des champs où la radioactivité du préparat se manifeste avec évidence surtout au niveau de la capsule des cloisons capsulaires et des nodules lymphatiques qui sont souvent entourés par une bande épaisse de noircissement à cause d'une particulière accumulation du radi-isotope au niveau de leur région périphérique (fig. 3). Ce tableau est plus accentué lorsqu'il y a dans le ganglion une hyperplasie réactive qui au point de vue morphologique ne présente pas de caractères de spécificité et d'éléments tels qu'on la puisse différencier d'une commune lymphadénite réactive spécifique (DeL Bo). Pour cette raison lorsque dans les ganglions



Fig. 2. Detail à plus fort agrandissement de la fig. 1. La gauche autoradiographie à durée coloration à 1 h/cm. (cos)

apparaissent des signes tout à fait initiaux de colonisation néoplasique (qu'on peut néanmoins documenter autoradiographiquement par une comparaison soigneuse avec le préparat coloré). Le tableau du préparat autoradiographique dans son ensemble ne diffère pas de celui qu'on peut observer lorsque dans le ganglion prévalent, en l'absence de micrométastases, les phénomènes réactionnels de type productif avec une prolifération marquée des éléments réticulo-endothéliaux des sinus intermédiaires.

Au contraire, la radioactivité dénotée par un noircissement intense et intégral au niveau des îlots de cellules néoplasiques devient imposante lorsque le parenchyme lymphatique est envahi par une colonisation métastatique massive. Dans ces cas le tableau autoradiographique ressemble à celui qu'on peut observer dans les autoradiographies des tumeurs laryngées qui ont produit les métastases lymphoglandulaires (fig. 4).

Dans les ganglions lymphatiques de même que dans les tumeurs laryngées, la radioactivité, dénotée par les autoradiographies, est moins accentuée dans les foyers de nécrose et fusion néoplasique dans les agglomérats de cellules épithélio-mateuses hyperkératinisantes ou lorsqu'on avait fait auparavant de la roentgentherapie.

CONCLUSIONS

Une radioactivité exalte des îlots de tissu carcinomateux par rapport au tissu qui n'est pas atteint par le processus néoplasique est particulièrement ex-

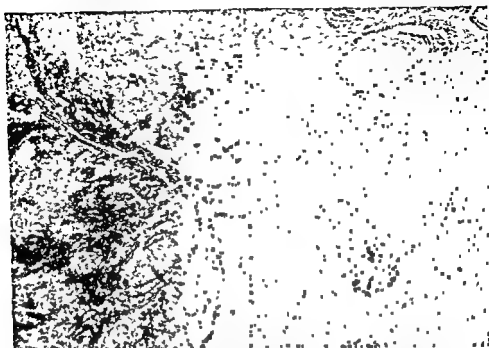


FIG. 3. Detail d'un fragment de nodule lymphatique sans métastase. *À gauche*, autoradiographie, *à droite* coloration à l'hématoxyline. La fixation du P^{32} est évidente au niveau des cloisons capsulaires et des follicules lymphatiques.

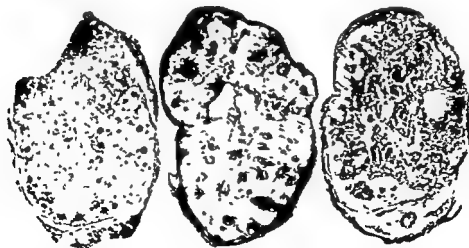


FIG. 4. Vue générale de deux nodules lymphatiques hyperplastiques et d'un nodule lymphatique métastatique. *À gauche*, autoradiographie d'un nodule avec de l'hyperplasie peu accentuée. *À milieu*, autoradiographie d'un nodule avec de l'hyperplasie plus accentuée. *À droite*, autoradiographie d'un nodule massivement envahi par la colonisation néoplasique. On doit souligner la différence de fixation du P^{32} par les deux nodules lymphatiques hyperplastiques et par le nodule métastatique.

dente à l'autoradiographie, lorsque l'examen est porté sur les carcinomes laryngés et sur ces ganglions lymphatiques satellites dans lesquels la colonisation métastatique est imposante et massive. Lorsque la colonisation métastatique est initiale ou peu étendue les dots limites de tissu néoplasique existent. C'est vrai une radioactivité élevée, mais le tableau autoradiographique dans son ensemble n'est pas suffisamment démonstratif car des autres segments du ganglion expliquent un degré considérable de radioactivité, surtout lorsqu'il y a des phénomènes marqués d'hyperplasie réactionnelle réticulo-endothéliale.

Les résultats de nos recherches sont en concordance avec les connaissances actuelles sur le pouvoir de fixation du P^{32} par ces tissus qui comme les néoplasiques se trouvent en état de métabolisme exalté et de croissance plus rapide et tumultueuse. De plus ils expliquent les résultats que nous avons obtenus précédemment et qui démontraient que la radioactivité mesurée par un détecteur de Geiger Muller sur le tissu néoplasique homogénéisé était remarquablement plus élevée que celle du tissu normal environnant.

Par les recherches actuelles il est donc documenté d'une manière précise et objective que la plus grande radioactivité des régions du larynx qui sont siège d'un carcinome est due effectivement à une particulière activité de fixation et d'accumulation du radio-isotope par les cellules néoplasiques.

Il nous semble que les résultats obtenus peuvent représenter un point de départ pour une éventuelle application clinique de la méthode des radio-isotopes comme moyen complémentaire et subsidiaire pour le diagnostic des tumeurs du larynx et des voies aéro-digestives en général.

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I Prof. E. Pallistrini e P. Filippi della Vittoria 6 Gènes

DISCUSSION

A. Eckert Mubius. Die schonen Untersuchungen Pallistrinis erscheinen mir als ein wesentlicher Beitrag zur Feststellung, ob Lymphknotenschwellungen carcinomverdächtig sind oder nicht. Für die einzuschlagende Therapie ist dies von entscheidender Bedeutung. Fast mehr noch aber interessiert uns die Frage, ob mit dieser

Methode auch kleine Tumoren des Kehlkopfes festzustellen sind die laryngoskopisch schlecht diagnostiziert werden können. Es gilt dies vor allem für die aus der Tiefe des Ventrikels hervorgehenden Carcinome, die vielfach sich nach dem „Fettkörper“ zu entwickeln, ohne autoskopisch, endoskopisch und probediagnostisch gesichert werden zu können, wenn man nicht aus dem Fettkörper direkt von aussen oder innen eine Probeexcision entnimmt. Gar nicht selten muss in solchen Fällen ohne positive Probeexcisionsergebnis aus rein klinischer Diagnose der operative Eingriff vorgenommen werden. Es wäre sehr vorteilhaft, wenn man solche beginnenden Tumoren mit P³² feststellen könnte und ich bitte um Auskunft, ob auch bei so kleinen Tumoren diese diagnostische Möglichkeit vorliegt.

E. Pallesstrini (Reponse) La méthode proposée ne peut servir pour un diagnostic précoce parce que, comme j'ai démontré, il faut que la néoplasie soit bien développée pour la distinguer d'une lésion inflammatoire. C'est pour cela que j'avais dit que c'est un moyen diagnostique complémentaire et subsidiaire.

Pour le diagnostic précoce j'ai fait construire un enregistreur de Geiger Muller long de 27 cm et subtil pour l'employer dans les laryngoscopies directes. La fragilité et le coût des appareils ont limité mes observations aux 20 cas cliniques. Je peux dire que dans 2 de ces cas on a pu faire un diagnostic précoce d'un cancer du ventricule de Morgagni qui à l'endoscopie laryngienne indirecte n'avait pas été visible parce qu'il avait été couvert de la bande vocale supérieure. Il faut poursuivre la recherche pour la mieux affirmer.

RECHERCHES SUR LE TRAUMATISME SONORE LABYRINTHIQUE

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Les travaux de McCabe et Lawrence (1956) ont montré que des animaux soumis, pendant 20 min à des bruits intenses composés de fréquences allant de 1000 à 10000 c/s présentent, en plus des lésions cochléaires bien connues, des altérations histologiques du saccule. Par ailleurs, une publication toute récente de Mangabeira-Albernaz, Covei et Eldredge (1959) fait état d'altérations histologiques, non seulement du saccule, mais aussi de l'utricule chez le cobaye soumis à des sons purs variant de 170 à 20000 c/s et d'intensités supérieures à 140 db.

Il apparaît ainsi que les traumatismes sonores ne sont pas l'appanage exclusif de la cochlée mais également du vestibule.

Nous avons de notre côté recherché si des stimulations sonores intenses et prolongées ont un effet traumatisant sur la fonction des canaux semi-circulaires.

Pour ce faire nous nous sommes servis du Pigeon qui offre la grande facilité de permettre l'enregistrement du nystagmus per et post rotatoire *cephalique* caractéristique de cet animal grâce à la dérivation par deux paires d'aiguilles des électrodes des électromyogrammes des deux masses musculaires gauche et droite de la nuque (voir van Eick, 1953).

Les expériences ont été poursuivies de la façon suivante. L'animal non anesthésié dont la tête est parfaitement fixée par une tière adéquate dans la position favorable à la stimulation rotatoire des canaux semi-circulaires horizontaux est soumis dans l'obscurité totale à une rotation horaire de trois tours en 15 sec. La fig. 1 A₁ montre les électromyogrammes per et post rotatoires obtenus dans ces conditions.

Ensuite, après cet enregistrement préliminaire, l'animal est soumis pendant 60 min à une stimulation par un son pur intense (700 c/s à 100 db dans l'expérience illustrée par la fig. 1) grâce à un tube de caoutchouc adapté au mur auditif gauche. Nous insistons sur le fait que l'animal ne subit aucun artifice de préparation facilitant l'atteinte des crêtes ampullaires par les vibrations sonores et en particulier pas de fenestration du canal semi-circulaire ne cessant de provoquer le phénomène de Tullio.

Dans le but d'observer les effets éventuels de cette stimulation sonore de l'oreille gauche sur la fonction du canal horizontal, l'animal est soumis immédiatement après à une stimulation rotatoire des canaux semi-circulaires horizontaux (trois tours en 15 sec). L'électromyogramme correspondant montre d'une façon particulièrement évidente (fig. 1 A₂) que le nystagmus postrotatoire

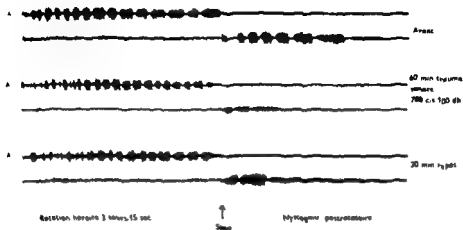


Fig. 1

toire (résultant de la stimulation de l'ampoule horizontale gauche) est très fortement réduit en amplitude et en durée.

Si ensuite l'animal est laissé au repos on assiste, dès les premières minutes, à une récupération progressive du nystagmus altéré. Le tracé de la fig. 1₂ montre que cette récupération est déjà très importante après 20 min de repos.

Il paraît donc ainsi démontré que chez le pigeon n'a eu lieu aucune adaptation. La stimulation d'une oreille par un son intense et prolongé est susceptible de réduire considérablement la sensibilité de la crête sensorielle d'un canal semi-circulaire.

Le nombre relatif (un grand de pigeons (une vingtaine) que nous avons soumis à des traumatismes sonores divers — et ne pûmes de fréquences variant de 100 c/s à 2000 c/s — chocs ou bruit blanc — nous permet de dire qu'il existe, comme pour la cochlée, le saccule ou l'utricule, une susceptibilité très variable des canaux semi-circulaires aux traumatismes sonores. Pour une même stimulation de même intensité et de même durée, tel animal accusera une altération définitive de la réponse à la rotation, tel autre une altération totalement réversible et tel autre encore pas d'altération du tout.

On peut se demander par quel mécanisme les vibrations sonores atteignent la crête impulsive dans un canal non fenestré (il ne s'agit pas d'un phénomène de Füllig). Est-ce par l'intermédiaire du vestibule ou s'agit-il d'une altération par transmission osseuse d'un son dont l'intensité mettrait tout le crâne en vibration? Cette dernière hypothèse ne nous semble pas être valable. La lésion crânienne du pigeon se prêtant mal, par sa pneumatisation extrême, à une telle propagation, et par ailleurs, dans une telle hypothèse, nous devrions assister à une altération similaire du labyrinthe opposé, ce qui n'est pas le cas.

On sait d'autre part que Bocca et Perini ont tout récemment (1960) remis en question — en se basant sur des observations cliniques, la possibilité d'une participation vestibulaire (sacculaire) à la fonction auditive. Des lors le fait qu'un canal semi-circulaire est susceptible de subir un traumatisme sonore permet-il de conclure à une fonction auditive de la crête ampullaire? Nous ne pensons pas que cette hypothèse doive être retenue. Les intensités sonores auxquelles le canal semi-circulaire est sensible dans nos expériences étant trop élevées pour être considérées comme « physiologiques ».

Il semble des lors raisonnable d'admettre que l'action mécanique des ondes sonores — nécessairement de fortes intensités — est transmise aux crêtes ampullaires par l'intermédiaire du liquide utriculaire dont elles sont si voisines. La mise en évidence d'un traumatisme sonore utriculaire (Mangaburn, Albermar et Covell) vient à l'appui de cette conception.

RÉSUMÉ

Chez un pigeon non préparé n'ayant en particulier pas subi une fenestration du canal semi-circulaire nécessaire au déclenchement du phénomène de lullio la stimulation prolongée (60 min) d'une oreille par un son de forte intensité (100 db) est capable de provoquer un « traumatisme sonore labyrinthique » s'ajoutant au traumatisme sonore cochléaire. Ce traumatisme sonore labyrinthique se caractérise par une forte réduction de l'amplitude et de la durée du nystagmus réactionnel à une stimulation rotatoire. Ces altérations peuvent être réversibles. Leur mécanisme pathologique est décrit.

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DISCUSSION

M. van Läck (Réponse). Pour admettre l'hypothèse soulevée par Henneclart d'un réflexe inhibiteur central responsable des altérations des réflexes des canaux semi-circulaires à la suite d'une stimulation sonore de l'oreille il faudrait que la réversibilité du phénomène soit beaucoup plus rapide qu'elle n'est. On n'expliquerait pas non plus les cas où il n'y a pas de retour à une réponse améliorée comme je l'ai observé chez l'animal et comme le Prof. Ferrari qui a eu l'amabilité de prendre la parole à sa vue chez l'homme.

Par ailleurs les expérimentateurs américains comme Covell ont trouvé des altérations histologiques importantes du saccule et de l'utricule.

THE INFLUENCE OF SOME DRUGS UPON THE FUNCTION OF THE LABYRINTH

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Some simple techniques are described by which the efficacy of anti seasickness drugs as depressors of vestibular function can be investigated by judging the effect on the recording of the eye movements by electronystagmography.

A simple rotatory test informs us about the cupula organ. The parallel swing indicates the function of the otolith organ as is shown in a series of patients with total and partial loss of labyrinthine function.

Since electronystagmography (E.N.G.) has become a reliable method of registering the movements of the eyes during vestibular stimulation, it has also become possible to express the results of such a stimulation in objective numbers. This objective reflection in numbers has been used before in the duration of various answers to caloric and rotatory stimuli but this appears to be only apparent accuracy. Electronystagmography proved that the speed of the slow phase of nystagmus is a much better indicator for the vestibular answer than its duration both in caloric and rotatory stimulation. I do not intend to go into the details of the technique of electronystagmography or into the difficulties which accompany the calibration of the whole set up from the amplifier to the test person's eye movements. I have gone into these factors at length at another occasion (Jongkees-Hamersma).

My assistant Dr. Philipszoon has used E.N.G. to study the effect of some anti seasickness drugs upon the labyrinth. As it seems reasonably certain that the stimulated vestibular organ is a necessary link in the development of motion sickness, the effect of a number of these drugs on vestibular reaction was examined.

The first aim of the investigation was to find an easy and easily reproducible technique of recording the reactions after vestibular stimulation and the effect of drugs upon these reactions.

The first series of examinations was aimed at the cupula function. As a small rotatory stimulus already provokes a distinct nystagmus in rabbits, it was sufficient to cause a rotation over a small angle. During the experiment the rabbit was kept in darkness in a box to avoid changes in the corneal retinal potentials. The electrodes were fixed in front of the eye and behind it. An identical rotation was the stimulus in all our experiments. In order to calibrate the eye movements of a rabbit, a special difficulty had to be surmounted. As we cannot ask the animal to look at some point, we had to move its eye by

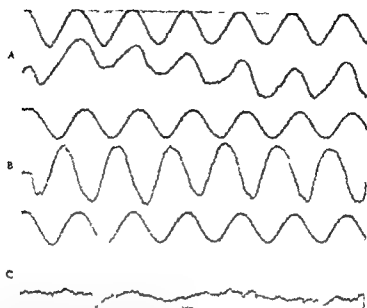


FIG. 3. Lye movements of human beings on the parallel swing (a) normal test person (b) patient after unilateral labyrinthectomy (c) patient with two dead labyrinths. Upper curve, movement of the swing; lower curve, eye movement.

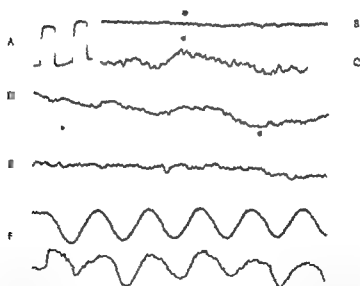


FIG. 4. Eye movements of a patient after bilateral fenestration (a) calibration 20 (b) rotatory stimulation in the plane of the horizontal canals (stop indicated by dot, 60°/sec) (c) rotatory stimulation in the plane of the vertical canals (stop indicated by dot, 60°/sec) (d) fistula test (first dot positive pressure, second dot negative pressure) (e) caloric test with ethyl chloride (f) parallel swing test.

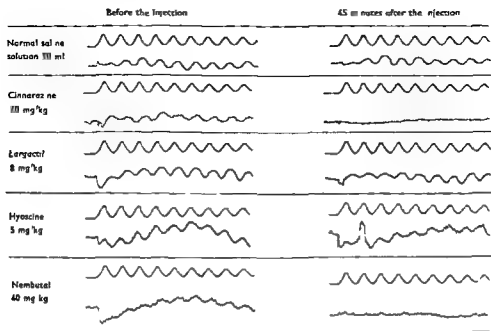


FIG. 5. The effect of various drugs upon the eye movements of rabbits on the parallel swing. Upper line: movement of the swing; lower line: movements of the eyes.

in the form of a sine. Here we find a clear reaction of the position of the eye to linear accelerations and an identical influence of the examined drugs. As the parallel swing test is much simpler and smoother and as the lift test did not increase our amount of reliable data, we prefer the extremely easy and cheap parallel swing.

In clinical experiments with the aid of cupulometry and parallel swing test all our findings in the rabbit could be corroborated (Philipszoon).

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DISCUSSION

J. Tachmann. I would like to add some words to the excellent communication of Prof. Jongkees. I have made more than 60 experiments with intravenous injections of various drugs. Laryacetil had a facilitatory influence on the nystagmus, elicited by electrical stimulation at the n. a. There was no latency, augmentation of the frequency of beats, lowering of the threshold and very great prolongation of after

nystagmus up to 3.51, whereas it stopped the equilibrium disturbances due to unilateral labyrinthectomy. Tigan Roche, on the other hand, had had an inhibitory influence on the central nystagmus, but no influence at all on equilibrium disturbances. Pentothal 6 mg/kg i.v. had facilitatory influence on central nystagmus (after nystagmus of 14°) and stopped equilibrium disturbances after unilateral labyrinthectomy.

From those observations we are entitled to suggest that the source of origin of nystagmus is not connected with the source of origin of equilibrium disturbances due to unilateral labyrinthectomy.

A. Meyer zum Göttesberge: Ich möchte bei Gelegenheit des interessanten Vortrags von Dr. Jongkees auf die Medikamente hinweisen welche Nystagmus provozieren. Ich erwähne das Morphin, der Vertikalnystagmus nach unten und das Nicotin, das in 50% Vertikalnystagmus nach oben erzeugt. Schliesslich den Alkohol, Lagenystagmus, der einen regelmässigen phasischen Ablauf zeigt.

L. B. W. Jongkees (Réponse): Je voudrais répondre à Messieurs Lachmann et Meyer zum Göttesberge que j'ai seulement donné nos résultats de l'examen de l'organe vestibulaire, spécialement dans sa partie otolithaire. Je n'ai pas parlé des résultats cliniques ni de médicaments nystagmogenes.

ETUDE ELECTROVISTAGMOGRAPHIQUE DU SYNDROME DE MENIERE

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Clinique O R L de l'Université de Genève

Sur un matériel de 60 observations cliniques nous avons retenu 42 cas de Meniere typique exempts de toute maladie de l'oreille de traumatisme crânien ou d'autre affection neurologique dont les examens cliniques et les E \ G étaient complets et corrects (fig 1). La triade symptomatique vertige, hypoacoustie, acouphènes est complète dans la moitié de notre série dans l'autre moitié l'existence d'acouphènes n'a pas été signalée. La répartition est égale entre les deux sexes. L'âge des patients est compris entre 40 et 60 ans dans $\frac{2}{3}$ des cas.

Symptômes généraux

La prédisposition aux *migraines* en dehors des crises de vertiges a été notée dans 42%, dont sept fois accompagnées de céphalées violentes ou très violentes. Au contraire un *terrain allergique* caractérisé par une eosinophilie sanguine ou par une intolérance alimentaire n'est apparu de manière du reste peu caractéristique que chez $\frac{1}{3}$ des patients. La *pression artérielle* plutôt basse était en moyenne de 137-83 entre 20 et 40 ans et de 132-80 entre 40 et 60 ans avec plusieurs fois une hypotension pouvant atteindre 90-50 dans ce dernier groupe.

Inversement la *pression artérielle relâchée* minima était en moyenne de 40 gr au dessous de 40 ans et de 43 gr entre 40 ans et 60 ans soit légèrement plus élevée que normalement. Il y a donc bien une discordance entre P A et P A R discrète mais constante indiquant une dysrégulation vaso motrice cérébrale.

Audiométrie

Elle a révélé soit une *hypocoûse* « en plateau » très accusée (50%) fréquemment bilatérale et prédominant tantôt à droite tantôt à gauche soit une hypocoûse inférieure à 20 db de forme peu caractéristique (en cloche ou petit hiatus sur 4000 c/sec) et parfois réversible en quelques jours. Le *recrutement* a pu être mis en évidence 12 fois seulement (26%) par le test de Fowler, le test de l'uscher a été peu concluant. Des *acouphènes* ont été constatés dans 23 cas (51%) le plus souvent unilatéraux tantôt à droite tantôt à gauche.

Symptômes vestibulaires

Ils comprennent les vertiges, les signes objectifs et les symptômes mis en évidence par les épreuves thermiques ou rotatoires.

ENG 934

ÉPREUVE GIRATOIRE LIMINAIRE

SYNDROME DE MENIERE

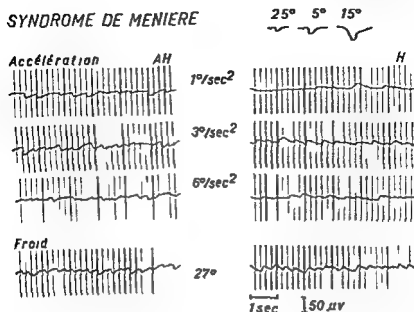


FIG. 2. Nystagmus réactionnel de petite amplitude invariable aux accélérations de $1^\circ/\text{sec}^2$ de $3^\circ/\text{sec}^2$ et de $6^\circ/\text{sec}^2$ dans le sens antihoraire (AH) comme dans le sens horaire (H). De même aux épreuves thermiques.

fois à droite et une fois à gauche. Toutefois dans six autres cas les réponses ont été en partie nulles ou masquées par des artefacts et d'interprétation difficile ou même impossible. Bien que nettement supérieur à l'observation directe, l'ENG a donc donné des renseignements valables que chez la moitié des patients examinés de cette manière.

Épreuve giratoire liminaire

Cette épreuve (déjà souvent décrite précédemment) comporte deux périodes d'accélération, décélération et une période d'accélération suivie d'un arrêt brusque. Les nystagmus per-rotatoires et post-rotatoires sont enregistrés dans l'obscurité (L.N.G.).

Les électro-nyctagmogrammes ainsi obtenus ont été divisés en deux groupes ayant des caractères communs et des caractères différents.

Les caractères communs à tous les syndromes de Menière sont :

- un nystagmus d'une amplitude petite ou très petite (1° à 3°) qui le distingue immédiatement et qui est invariable quelle que soit l'intensité de la stimulation (fig. 2)
- un seuil nystagmique légèrement élevé ($2^\circ/\text{sec}^2$ à $3^\circ/\text{sec}^2$) de chaque côté de manière symétrique souvent difficile à déterminer parfois variable et un raccourcissement correspondant de la durée du nystagmus post-rotatoire.

ENG 980

EPREUVE GIRATOIRE LIMINAIRE

SYNDROME DE MENIERE

25° 5° 15°

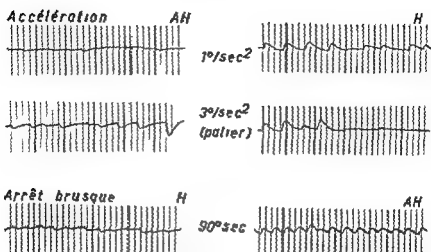


FIG 3 A une accélération de $1^\circ/\text{sec}^2$ on note une prédominance directionnelle à droite (sens horaire = H) et à $3^\circ/\text{sec}^2$ une prédominance à gauche (sens antihoraire = AH) qui se traduit par une durée plus longue de la période post stimulateur

Le premier groupe (type I) comprend 26 cas (58%) dans lesquels une prédominance directionnelle (PD) a été constatée

Chez 17 patients (38%) dont sept avaient un nystagmus spontané une prédominance directionnelle unilatérale ou prédominance unidirectionnelle s'est manifestée à l'ENG lors de l'épreuve giratoire liminaire 14 fois à droite et trois fois à gauche (au lieu de dix fois seulement aux épreuves thermiques avec ou sans enregistrement). Il a été possible en outre de préciser l'intensité du stimulus giratoire nécessaire à surmonter la PD chez 15 patients soit deux fois à $3^\circ/\text{sec}^2$ et cinq fois à $6^\circ/\text{sec}^2$. La direction du nystagmus giratoire prédominant fut toujours la même que celle du nystagmus thermique et du nystagmus spontané soit en général du côté opposé à l'oreille hyposcousique ou la plus sourde.

Chez neuf patients (20%) de curieuses particularités du nystagmus liminaire ou supraliminaire sont apparues soit sous la forme d'une prédominance alternant tantôt à droite tantôt à gauche soit sous la forme d'un *erethisme bilatéral et symétrique des réactions nystagmiques*. Ainsi dans l'ENG n° 980 on observe une prédominance directionnelle à droite au seuil à $1^\circ/\text{sec}^2$ puis une prédominance à gauche à $3^\circ/\text{sec}^2$ et finalement une durée prolongée du nystagmus dans les deux directions. Dans l'ENG n° 1908 on a PD gauche à $1^\circ/\text{sec}^2$ PD droite à $6^\circ/\text{sec}^2$ et dans l'ENG n° 788 on constate une PD droite à $3^\circ/\text{sec}^2$ et une PD gauche à $6^\circ/\text{sec}^2$. Nous avons appelé *prédominance alternée* ce phénomène qui à notre connaissance n'a jusqu'ici pas été décrit et qui se rapproche du suivant (Fig 3 et 4).

ENG 980

ÉPREUVE GIRATOIRE LIMINAIRE

SYNDROME DE MENIERE

25° 5° 15°

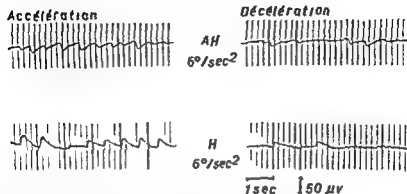


FIG. 4 (Même cas que fig. 3) A l'accélération antihorale (AH) à $6^\circ/\text{sec}^2$ le nystagmus gauche de la période d'accélération se prolonge pendant toute la durée du palier à vitesse constante (3 min) et empêche même la réaction de sens opposé à la décélération. La réaction de sens horaire (H) de même se prolonge pendant toute la durée du palier à vitesse constante et empêche la réaction de sens opposé à la période de décélération (crétisme bilatéral).

Dans quelques cas on observe une réaction d'embble excessive dont la durée augmente encore aux intensités supraliminaires de $3^\circ/\text{sec}^2$ et de $6^\circ/\text{sec}^2$, dans les deux directions soit une « prédominance bidirectionnelle » souvent symétrique (ENG 1084 1124 897 974) témoignant d'un *crétisme nystagmique* bilatéral avec ou sans alternance.

Ces faits suggèrent l'existence d'un *crétisme* et d'une instabilité des centres nystagmogènes plutôt que des récepteurs périphériques.

Le deuxième groupe (type II) comprend 19 cas (42%) qui présentent une remarquable uniformité.

L'amplitude des nystagmus réactionnels, per-rotatoires, post-rotatoires et thermiques est ici *extraordinairement* petite (1° à 2°) d'une valeur nettement inférieure à l'amplitude minima que l'on observe chez les sujets normaux ou dans les affections vestibulaires périphériques.

La fréquence est dans l'ensemble un peu plus rapide que d'ordinaire, située entre 2 et 4 secousses au lieu de 1 à 3 par seconde. Contrairement à l'amplitude elle augmente légèrement aux intensités supraliminaires d'accélération.

Le seul nystagmique souvent difficile à évaluer est en général symétrique pour les deux oreilles. Il est presque normal ou modérément élevé à $3^\circ/\text{sec}^2$ rarement situé plus haut. De même la durée des nystagmus post-rotatoires est faiblement diminuée de manière symétrique.

Enfin on observe fréquemment à des stimulations supra-liminaires une véritable *inhibition* qui se manifeste par une disparition partielle ou complète momentanée ou durable de la réaction nystagmique per- ou post-rotatoire.

ENG 934

EPREUVE GIRATOIRE LIMINAIRE

SYNDROME DE MENIERE

25° 5° 15°

Arrêt brusque 90°/sec

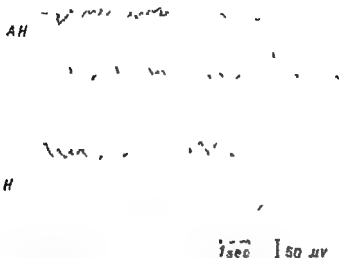


Fig 5 Inhibition du nystagmus post rotatoire après un arrêt brusque dans le sens antihoraire (AH) qui se traduit par une pause de 4 sec. De même dans le sens horaire (H) après un arrêt brusque également qui se traduit par une pause de 12 sec.

(fig 5). Très caractéristique aussi est l'« effacement » ou la disparition de la réaction pendant la stimulation et sa réapparition avec une amplitude croissante après la fin de celle-ci. La durée de la réaction post stimulatoire n'est pas diminuée mais au contraire augmentée par une sorte d'excitisme réactionnel.

Toutes ces particularités indiquent qu'il s'agit bien d'un phénomène d'inhibition centrale qui influence principalement l'amplitude déjà très petite des réactions nystagmiques plutôt que d'une diminution de la sensibilité des récepteurs vestibulaires. On peut également observer le phénomène inverse de « libération » du nystagmus dont l'amplitude augmente soudain de manière notable.

RÉSUMÉ

Dans 15 cas de Ménière typique une étude comparative et statistique des symptômes cliniques des épreuves thermiques et de l'épreuve giratoire liminaire montre que cette dernière a donné des informations beaucoup plus complètes et plus précises mais concordantes avec celles des deux autres méthodes. L'analyse des documents électro-nystagmographiques (ENG) a mis en évidence les principaux caractères suivants :

- une amplitude très petite (1-2°) du nystagmus à toutes les stimulations
- une fréquence relativement élevée (2-4 secousses par seconde)
- un seuil nystagmique légèrement élevé sans rapport avec le seuil d'audition instable et souvent difficile à évaluer.

— une prédominance directionnelle (type I = 58 %) qui peut dans certains cas alterner d'un côté à l'autre suivant l'intensité du stimulus

— des phénomènes d'inhibition (type II = 42 %) sous forme d'une réduction globale de l'amplitude du nystagmus (à 1° ou moins encore), et de périodes de latence nystagmique per stimulatoires ou post stimulatoires

Ces différentes particularités suggèrent l'idée d'une forte participation des voies vestibulaires centrales et même vraisemblablement d'un trouble vaso-moteur initial de certaines structures méso-diencephaliques les lésions de l'oreille interne n'étant que secondaires

ZUSAMMENFASSUNG

In 45 typischen Fällen von Ménièrescher Krankheit zeigt eine vergleichende und statistische Untersuchung der klinischen Symptome, der kalorischen Nystagmusprüfung und der Drehschwellenprüfung nach Montandon, dass diese letztere viel vollständigere und genauere, aber mit den zwei anderen Methoden übereinstimmende Auskunft gibt. Die Analyse der elektronystagmographischen Dokumente hat folgende hauptsächlichste Charakteristika gezeigt:

- 1) Eine sehr kleine Amplitude (1–2.5) des Nystagmus für alle Reizungen
- 2) Eine verhältnismässig hohe Frequenz (2–4 Schläge pro Sekunde)
- 3) Einen leicht erhöhten Nystagmusschwellenwert, welcher wechselnd und häufig schwierig bewertbar und ohne Beziehung zum Gehörschwellenwert ist
- 4) Eine richtungsgebundene Nystagmusbereitschaft (Typus I = 58 %) die in gewissen Fällen, je nach Intensität des Reizes, von einer Seite zur anderen wechseln kann
- 5) Hemmungserscheinungen (typus II = 42 %) in Form einer globalen Verkleinerung der Nystagmusamplitude (bis zu 1° oder sogar weniger) und die Erscheinung von Latenzperioden während oder nach der Stimulierung

Diese verschiedenen Eigentümlichkeiten legen die Idee einer starken Beteiligung der zentralen Vestibularisbahnen und wahrscheinlich sogar einer ursächlichen vasomotorischen Störung von gewissen mesodiencephalen Strukturen, wobei die Innenohrveränderungen nur sekundärer Natur sind, nahe.

SUMMARY

In 45 cases of typical Ménière's disease a comparative and statistical study of the clinical symptoms of the results of the caloric tests and of the *liminal rotatory test* shows that the latter has furnished much more complete and precise information which is in agreement with the two other methods. The analysis of the nystagmic records (E.N.G.) reveals the following characteristics:

- 1) A very small amplitude (1–2.5) of the nystagmic reaction after every stimulation
- 2) A relatively high frequency (2–4 jerks per second)
- 3) A slight elevation of the nystagmic threshold which is not proportional to the auditory threshold is unstable and often difficult to evaluate
- 4) A directional preponderance (type I = 58 %) which alternate in certain cases from one side to the other depending upon the intensity of the stimulus
- 5) Phenomena of inhibition (type II = 42 %) in the form of an overall diminution of the amplitude of the nystagmus (1° or even less) and periods of nystagmic per-rotatory or post-rotatory latency

These different particularities suggest an important participation of the central vestibular pathways and probably of an initial disturbance of vaso motor origin in some mesodiencephalic structures, the lesions of the internal ear being subsequent only.

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MUTUAL INFLUENCE OF NYSTAGMOGENIC CENTERS DURING LABYRINTHINE OR CENTRAL NYSTAGMUS

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Unilateral electrical stimulation at the nystagmogenic area (n_1) of the rabbit is accompanied by the following phenomena

- 1 The electrocorticogram does not reflect any systematic changes
- The contralateral n_2 does not participate in the nystagmus reaction
- 3 The vestibular nuclei record faithfully the frequency of the nystagmogram however, the action potentials increase with time until they reach their full amplitude The after nystagmus observed after discontinuation of the stimulation is also reflected in the electrogram of the vestibular nuclei Likewise the latter records exactly all forms of vestibular nystagmus Thus the vestibular nuclei appear to be at least in part responsible for the nystagmus rhythm

Previous experiments on central nystagmus produced by electrical stimulation of the nystagmogenic area (n_1) in the meso diencephalon of the rabbit have demonstrated the functional interaction of the peripheral vestibular apparatus and the vestibular nuclear complex in the medulla with the n_2 Our conclusions were based on the superposition of labyrinthine and central nystagmus and on the results of transection experiments (1-3)

In the present investigation this relationship was studied by means of electroencephalographic recordings For stimulation or recording at the n_2 electrodes were placed in position bD bF (distance of the electrode tip from the upper surface of the socket was 12-14 mm) (Figs 1-2)

The vestibular nuclei were reached by electrodes at n_1H at a depth of 20-22 mm Recording from the temporo parietal cortex was achieved by epidural subarachnoid electrodes introduced at n_1H and at n_1F (Figs 1,3)

In Fig. 3 we show the nystagmogram obtained by electrical stimulation at the n_1 It is easily recognised that after an initial latency of a few seconds the nystagmic movements increase both in frequency and amplitude until they reach a certain maximum Finally after cessation of the stimulus a decaying after nystagmus appears

The results of the encephalographic recordings will be presented according to the specific stimulus applied

1 Rotation

In the majority of cases the n_2 reveals no change at all Rarely it shows increased general activity without any relationship to the nystagmus rhythm

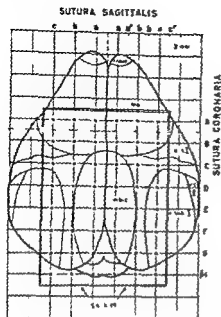


FIG 1

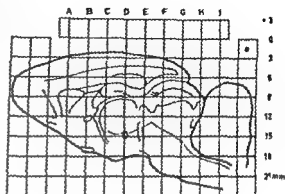


FIG 2

FIG 1 Stereotaxic socket for electrical stimulation of the rabbit's brain (atlas of Monnier & Gangloff)

FIG 2 Sagittal section through the rabbit's brain. Rectangle on top represents schematically the stereotaxic socket

However, when in the resting state left and right *n.a.* exhibited unequal EEG records the latter became markedly synchronised during pre- and post-rotatory nystagmus.

Similarly, in the cortical region, only unspecific signs of stress or attention can be discovered, i.e. the normal (resting) rhythm is replaced by waves of higher frequency and lower amplitude (Fig. 4).

2. Calorisation

Caloric nystagmus produces essentially the same changes as rotation.

3. Stimulation at the nystagmogenic area

As a rule the contra-lateral *n.a.* does not cooperate; sometimes it shows pronounced disorder. In single cases a conspicuous change was observed about 20 seconds after cessation of the after-nystagmus. The record showed signs of an attack of petit mal. Such reactions were entirely absent from the cortex or the nuclear complex (Fig. 5). Attacks after stopping electrical stimulation in the diencephalon (not related to central nystagmus) were reported by Petsche & Monnier (4) who interpreted them as rhinencephalic attacks.

The nuclear complex records the nystagmogram faithfully and the intensity of its waves corresponds to the intensity of the nystagmic movements. However, there is a clear cut delay in the start of the potentials as compared with



FIG 3 Nystagmogram of central nystagmus. Animal was stimulated at left b/D (Fig 1). Pulse characteristics: 20 c/s, 1.5 V, 2 msec duration. At signal (first arrow) start of stimulation. Latency: 7 sec, period of maximal constant frequency at b. At signal (second arrow) stimulation stops. After-nystagmus, during 11 sec, frequency of beats increases to 4 per sec. Cessation of after-nystagmus after 7 sec (c).

the nystagmogram. In the beginning, the potentials are slow and increase with time until they reach full amplitude. The after-nystagmus likewise is recorded by the medullar electrode. Similarly, spontaneous nystagmus can be recorded from the nuclear complex (Fig 6).

The cortex usually does not reflect the events of central nystagmus, very seldom spikes may be seen in the cortical potentials.

4 Stimulation at the vestibular nuclei (n_1H)

In many cases, introduction of an electrode into this region already causes spontaneous nystagmus, which is usually conjugated. But disconjugated eye movements have occasionally been observed.

When spontaneous nystagmus was absent after introduction of the medullar electrodes, nystagmus could be elicited by electrical stimulation. The response

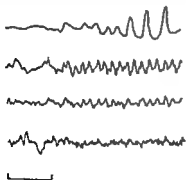


FIG 4

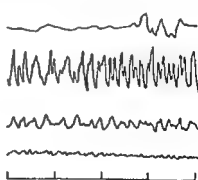


FIG 5

FIG 4 Postrotatory nystagmus. First row: right eye. Second row: potentials from right nystagmogenic area (n_1). Third row: potentials from left n_1 . Fourth row: potentials from temporo-parietal cortex. Time signal: 1 sec.

FIG 5 Signs of attack of petit mal. 20 sec after cessation of after-nystagmus. First row: right eye. Second row: n_1 . Third row: nuclear region (medulla oblongata). Fourth row: temporo-parietal cortex. Time signal: 10 sec.

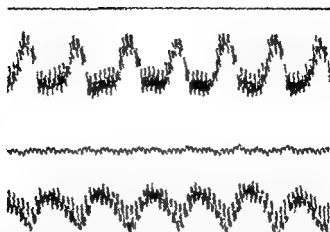


FIG. 6. Registration at stimulation at left *n.a.* (central nystagmus). First row: nystagmus right eye. Second row: potentials from right *n.a.* Third row: potentials from nuclear vestibular complex (medulla oblongata).

is almost indifferent to a change in frequency (from 2 c/s to 20 c/s) in contrast to the behavior of the *n.a.* The response as a rule is horizontal ipsilateral or contralateral conjugated nystagmus. Sometimes especially when the electrodes were pushed further in the form and duration of the nystagmus changed. It became rotatory, vertical or oblique accompanied by turning of the head.

During the stimulation of *n.H.* the contralateral *n.a.* showed a more pronounced increase of activity than the ipsilateral side.

The contralateral nuclear complex does not participate in the nystagmus recording during electrical stimulation of the other side but after cessation of the stimulation it does record the after nystagmus.

DISCUSSION

It is well known that the nuclear region in the medulla is affected by vestibular stimulation. This has been proved by various authors by direct recording from this region during stimulation of the labyrinth by rotation or caloric stimulation (2, 7). However, there is no specific anatomical representation in the vestibular nuclei for the various components of the peripheral labyrinthine apparatus. Lorente de No (8, 9) ascribes only to the medial triangular nucleus (Schwalbe) a higher associative function. This conception has recently found support in the findings of Pompeiano & Walberg (10) that fibers descending from the nucleus interstitialis (Cajal) through medial longitudinal bundle terminate only at Schwalbe's nucleus.

The phenomena of latency and after nystagmus are usually interpreted by the assumption that there exist several neuronal complexes representing a reverberating circuit which increases the potentials progressively above a critical threshold.

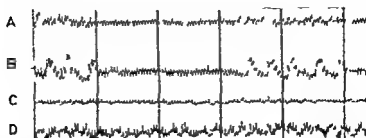


FIG 7 Stimulation at left *n. a.* after section from *a/II* to *b/II* (Fig 1) bilaterally \pm g of the vestibular nuclear complex produces constant potentials at the nuclear complex but inconstant nystagmus A central nystagmus left eye (interrupted during continuous stimulation) B central nystagmus right eye (interrupted during continuous stimulation) C right *n. a.* (potentials during continuous stimulation not changed) D vestibular nuclear region (medulla oblongata) (potentials during continuous stimulation not changed)

The same explanation may be used to explain the curious observation that the synchronous potentials in the vestibular nuclei lag behind the eye movements during central nystagmus. It is thus probable that at least one neuronal unit is intercalated between vestibular nuclei and the nystagmogenic area.

These findings also suggest that the vestibular nuclei are not the primary center determining the rhythm of nystagmus although they are an essential part of the nystagmus circuit. It should be recalled that Kempinski & Ward (11) demonstrated the activation of the lateral reticular formation by potentials ascending from the vestibular nuclei.

The vestibular nuclei play also an important role in the mechanism of central nystagmus. This is evidenced by the abolition of latency and after nystagmus when the connection between the nuclei and the posterior mesencephalon is severed by transection (3) at *G* as well as by destruction of the nuclear region at *H a-b* bilaterally and by the observation that this destruction in other cases may produce interruption of the central nystagmus during continued stimulation or sudden reversal of its direction as shown in Fig 7.

It is clear that also during labyrinthine nystagmus where the stimuli arrive first at the vestibular nuclei nystagmus can appear only after the center in the reticular formation has been brought into action. Latency during rotation is of the same magnitude as in central nystagmus suggesting that a comparable number of intermediate stations is involved in both cases.

The region of the vestibular nuclei represents a second area from which nystagmus can be elicited by electrical stimulation. It differs however from the *n. a.* characteristically in that (1) the nuclear complex responds to much lower frequencies than the *n. a.* and (2) the direction of the nystagmus response is not as clearly predetermined as in the *n. a.* This corresponds to the above statement that there does not exist a specific anatomical representation in this region. It is sometimes sufficient just to introduce electrodes into the nuclear region in order to provoke spontaneous conjugated or disconjugated nystagmus. The sensitivity of this region is also demonstrated by the fact that small

changes in electrode position, such as are produced by slight pressure upon the electrode, may change the direction of electrically evoked nystagmus or even abolish it. This is not the case with electrodes in the *na* and *mn*, perhaps be related to the crowded arrangement of nuclei in the medulla.

RÉSUMÉ

1 Nystagmus, provoqué par stimulation labyrinthaire, n'a pas d'effet synchronisant sur les potentiels du cortex temporo pariétal ou de la *na* (aire nystagmogénique dans le mésodiencephale du lapin). Les noyaux vestibulaires recordent fidèlement les mouvements nystagmiques.

2 Stimulation électrique d'une *na* n'est pas suivie par réponse systématique dans l'autre *na* ou dans l'écorce (cortex). L'ensemble des noyaux vestibulaires, au contraire, recorde le nystagmus central restant en arrière au commencement, mais recordant plus tard les mouvements des yeux, aussi durant le after nystagmus.

3 Stimulation électrique des noyaux vestibulaires provoque des formes et des directions variées de nystagmus. La *na* ne recorde pas ce nystagmus, mais montre une activité augmentée.

4 Les expériences éprouvent que l'ensemble des noyaux vestibulaires jouent un rôle important dans le rythme de toutes les formes du nystagmus, mais qu'il n'est pas le seul facteur décisif.

ZUSAMMENFASSUNG

1 Nystagmus, der durch labyrinthäre Reizung hervorgerufen wird, hat keinen spezifischen synchronisierenden Einfluss auf die Potentiale der temporo parietalen Hirnrinde oder auf die *na* (nystagmogene Area im Meso Diencephalon des Kaninchens). Die Vestibulariskerne geben getreu die Augenbewegungen wieder.

2 Elektrische Reizung einer *na* ruft keine systematische Antwort in der anderen *na* oder in der Hirnrinde hervor. Der Komplex der Vestibulariskerne dagegen gibt den zentralen Nystagmus wieder, etwas verspätet beim Einsetzen der Bewegungen, oder dann getreu den Nystagmusbewegungen folgend bis in die Periode des After nystagmus.

3 Elektrische Reizung der Vestibulariskerne bewirkt Nystagmus, der verschiedenartige Formen und Richtungen haben kann. Die *na* gibt diesen Nystagmus nicht wieder, zeigt aber erhöhte Aktivität.

4 Die Experimente beweisen, dass der Komplex der Vestibulariskerne eine wichtige Rolle im Rhythmus von allen Nystagmusformen spielt, dass er aber nicht der alleinige entscheidende Faktor ist.

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CHANGEMENTS DE LA CRÉATINE ET DU GLYCOGÈNE DES MUSCLES APRÈS LA DESTRUCTION CHIRURGICALE DU LABYRINTHE

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Après la destruction chirurgicale du labyrinthe des lapins selon de Kleyn les muscles de la partie hétérolatérale du corps devenus hypotoniques en comparaison à ceux de la partie homolatérale montrent une augmentation de la concentration de la créatine et une diminution du glycogène

Ces résultats correspondent avec ceux de nos études expérimentales précédentes sur les relations entre les solides l'humidité et les concentrations des ions de Ca et de Mg entre les deux moitiés du corps

INTRODUCTION

L'étude du fonctionnement des organismes vivants est fondée sur l'examen des phénomènes biophysiques et biochimiques qui se produisent dans ces organismes. Les progrès accomplis en biochimie ces dernières années ont permis dans la plupart des cas une parfaite mise en rapport des phénomènes physiques et des réactions chimiques qui se produisent simultanément dans l'organisme.

Celui qui étudie les phénomènes biologiques doit donc tenir compte du fait que toute manifestation physiologique de la matière vivante est un résultat de réactions chimiques qui se produisent dans la cellule même et se repercutent sur le liquide environnant.

Partant de ce point de vue nous avons essayé depuis quelques années de découvrir si l'hypotonie passagère des muscles qui suit la destruction chirurgicale unilatérale du labyrinthe est un résultat de troubles chimiques produits dans le muscle et nous avons effectué plusieurs séries d'expériences dont nous publions les résultats au fur et à mesure de notre recherche (1).

Nous avons ainsi constaté 1° que les chronaxies (2) du nerf sciatique du membre hétérolatéral au labyrinthe détruit présentent une augmentation rigide et 2° que les muscles devenus hypotoniques de la partie du corps hétérolatérale au labyrinthe détruit présentent par rapport à ceux de la partie homolatérale des troubles du métabolisme. L'eau p_{ex} et les cendres des muscles (3) les ions magnésium et calcium (4) présentent des différences sensibles si l'on compare les muscles devenus hypotoniques à ceux non influencés par la destruction du labyrinthe.

Dans la suite de cette recherche expérimentale nous avons également constaté des troubles de la créatine et de ses dérivés dans les muscles de la partie du corps hétérolatérale au labyrinthe détruit en comparaison avec les muscles de la partie homolatérale du corps. Il a été prouvé que ces troubles ne concer-

ment pas seulement la creatine et ses dérivés mais aussi le glycogène et l'acide lactique

Des dosages des composés cités ci-dessus ont également été effectués sur les muscles des deux parties du corps d'animaux témoins. On a effectué en tout cinq expériences sur des lapins sains et quinze expériences sur des lapins dont l'un des labyrinthes venait d'être détruit chirurgicalement.

Les résultats de ces expériences font le sujet du présent travail dans lequel nous exposons : 1° la méthode expérimentale, 2° les expériences, 3° l'interprétation des expériences et 4° les conclusions qui en sont tirées.

PARTIE EXPÉRIMENTALE

Opération de destruction du labyrinthe et ablation des muscles de la jambe

Les expériences ont été effectuées sur lapins comme suit :

1° *Opération de destruction du labyrinthe* Après avoir découvert le labyrinthe par la méthode de de Klein (1) nous procédons à sa destruction au moyen d'une curette fine.

2° *Ablation des muscles de la jambe* Sept à dix jours après la destruction du labyrinthe nous sacrifions l'animal en coupant sa carotide commune et nous procédons immédiatement à l'ablation des muscles de la jambe. Aussi tôt après l'ablation on broie les muscles en présence d'acide trichloroacétique à température très basse pour empêcher la glycogénolyse : une partie de la pulpe obtenue sert immédiatement aux divers dosages.

3° *Dosage de la creatinine* Après déprotéinisation de la pulpe on applique la méthode Benedict et Behre pour doser la creatinine (6).

4° *Dosage de la creatine libre* On a choisi pour éloigner la protéine le procédé Folin et Wu (7) qui empêche le doublement de l'acide creatine phosphorique par la transformation de ce dernier en sel insoluble.

Après cette déprotéinisation on ajoute du $ZnCl_2$ pour transformer la creatine libre en creatinine que l'on dose ensuite suivant la méthode déjà citée de Benedict et Behre.

La différence entre le taux total de creatinine trouvé cette fois et la creatinine dosée auparavant représente la creatine transformée en creatinine. Après avoir calculé la teneur en creatinine correspondant à la creatine libre on n'a qu'à multiplier la différence par 1.16 pour avoir les taux correspondants de la creatine.

5° *Dosage de l'acide lactique* Pour le dosage de l'acide lactique on applique le procédé Goldschmidt et Mendel (8) sur une autre partie de la pulpe.

6° *Dosage du glycogène* (9-9a) Pour le dosage du glycogène une partie de la pulpe est traitée en milieu basique. On ajoute ensuite de l'alcool et du 1-acétone qui précipitent le glycogène. Le glycogène précipité est soumis à l'hydrolyse en milieu acide : ce qui donne du glucose que l'on dose par la méthode de Hagedorn (10). Le taux de glucose multiplié par 0.95 représente la teneur en glycogène.

latérale du corps une augmentation du taux de la créatinine, de la créatine libre de l'acide lactique et une diminution du taux du glycogène

2 Les changements observés présentent certaines analogies avec ceux qui se produisent au cours de la contraction musculaire et surtout pendant la fatigue Ceci nous conduit à penser que la destruction hétérolatérale du labyrinthe provoque des troubles du métabolisme des glucides du muscle troubles qui causent probablement l'hypotonie du muscle

3 Les troubles de la glycogénolyse dans le muscle provoquent des changements analogues à l'ionogramme du muscle d'après les résultats obtenus d'une autre recherche déjà accomplie

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L'ADAPTATION DU SYSTEME VESTIBULAIRE PERIPHERIQUE

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Les effets inverses observés dans les réponses nystagmiques à l'épreuve rotatoire et à l'épreuve calorique pourraient n'être que la manifestation d'une adaptation de l'organe sensoriel pendant un déplacement de la cupule de durée prolongée.

En effet la modification d'activité électrique par la stimulation prolongée de la cupule manifeste une tendance au retour vers le niveau d'activité spontanée malgré que les forces stimulantes persistent dans leur action sur la cupule.

INTRODUCTION

Hallpike & Hood (1953) ont étudié chez l'homme l'affaiblissement de la réponse (response decline) de la cupule du vestibule à l'application d'un stimulus constant et de durée prolongée.

Utilisant un stimulus test de valeur déterminée (4° sec/sec pendant 6 secondes) ils ont pu observer un raccourcissement considérable de la durée de la sensation rotatoire sous l'effet de ce test si le sujet a été soumis au préalable à une accélération de même sens de 2°/sec/sec pendant 10 secondes.

Ces auteurs arrivent à la conclusion qu'il s'agit ici d'un phénomène d'adaptation plutôt que d'une fatigue physiologique mais ils ne cachent point la difficulté extrême qu'il peut y avoir à dissocier clairement l'un de l'autre ces deux phénomènes en particulier en ce qui concerne le fonctionnement du système vestibulaire. Même au niveau de la cochlée où les recherches ont cependant été extrêmement poussées (Living & Littler 1935, Fowler 1936, Davis 1943, Ruedi & Lurser 1942, von Békésy 1947, Caussé & Chavasse 1947, Hood 1950, 1955, Gisselson 1960, etc.) la distinction reste difficile à établir entre fatigue et adaptation.

La fatigue d'un récepteur sensoriel est caractérisée par un affaiblissement de la réponse qui se développe relativement lentement s'accroît avec l'intensité du stimulus et progresse indéfiniment avec le temps. La récupération de cette fatigue est lente.

L'adaptation est caractérisée par un affaiblissement de la réponse très rapide au départ puis de plus en plus lent jusqu'à ce que soit atteint un niveau constant de réponse. Le temps mis par l'adaptation de l'organe cochléaire pour être complet pourrait être de trois minutes et demie d'après Derbyshire & Davis (1935) ainsi que d'après Hood (1950). La récupération du récepteur se fait de façon particulière : si capable de répondre par des potentiels de haute fréquence revient très rapidement après la cessation du stimulus mais

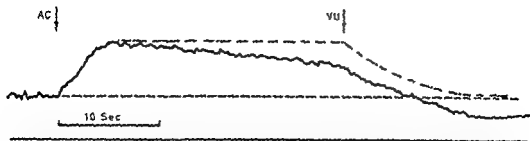


Fig. 2. Enregistrement au galvanomètre (trait plein) de l'activité du nerf abducteur pendant la durée d'une accélération prolongée (AC) de $5.6^\circ/\text{sec}^2$ et à la fin de cette accélération (vitesse uniforme = 1 U). Le trait — correspond au niveau d'activité prévue par la théorie.

Mais cette activité ne se maintient pas à un niveau constant pendant toute la durée de l'accélération. Elle s'affaiblit progressivement ainsi qu'on peut le constater sur la fig. 2. À la fin de l'accélération l'activité tend bien à rejoindre le niveau d'activité spontanée suivant la courbe exponentielle prévue par la théorie mais elle croise ce niveau en une longue phase d'activité réduite (effet inverse) ce qui laisse à penser que l'affaiblissement de la réponse se poursuit pendant toute l'évolution du mouvement du système tant que la cupule n'a pas retrouvé sa position de repos initiale.

Nous avons observé un phénomène semblable lorsque la cupule subit une force d'accélération intense mais de très brève durée (arrêt brusque) à la fin d'une rotation prolongée à vitesse uniforme.

L'activité subit une augmentation très rapide et importante (fig. 3) due à la brusque flexion de la cupule. Puis celle-ci reprenant sa position normale suivant la courbe exponentielle que prévoit la théorie l'activité diminue elle aussi mais plus rapidement croise le niveau de base et passe sous celui-ci pour une période prolongée avant de retrouver finalement sa valeur de départ.

La fig. 4 représente l'enregistrement de l'activité électrique du nerf abducteur droit au cours d'une expérience où se succèdent

a) une accélération de démarrage (D) l'activité subissant une inhibition totale brusque avec retour progressif vers le niveau de base puis une excitation modérée prolongée (effet inverse) pendant que se poursuit le mouvement rotatoire uniforme (vitesse $120^\circ/\text{sec}$ pendant 60 sec)

b) une accélération prolongée (4 s) de $4.9^\circ/\text{sec}^2$ l'activité subissant

dont la solution approximative est

$$\xi = \alpha \frac{\theta}{\Delta} (1 - e^{-\Delta t / \tau}) \quad (2)$$

Come chez la grenouille $\theta/\Delta = 0.026$ et $\Delta/\tau = 0.2$ l'équation (2) devient

$$\xi = 0.026 \alpha \left(1 - \frac{1}{e^{0.2t}} \right)$$

ce qui permet de déterminer pour chaque accélération α l'angle de flexion cupulaire ξ en fonction du temps t . Après un temps infini la flexion $t \rightarrow \infty = 0.026 \alpha$.



Fig. 3 Enregistrement au galvanomètre de l'activité du nerf ampullaire lors de l'arrêt brusque (1) d'une rotation prolongée à vitesse uniforme (120°/sec). Le trait — correspond au niveau d'activité prévu par la théorie.

une augmentation initiale puis s'affaiblissant progressivement pendant que se poursuit l'accélération.

c) l'arrêt de l'accélération (vitesse uniforme 120°/sec) l'activité s'effondrant sous la valeur de base et ne retrouvant cette valeur qu'après une minute environ.

d) une accélération prolongée de 48°/sec sec de même durée qu'en a) mais de sens inverse l'activité subissant une inhibition importante puis récupérant progressivement pendant que se poursuit l'accélération.

e) l'arrêt de cette accélération avec un croisement du niveau de base par l'activité qui s'excite rapidement pour ne retrouver le niveau initial qu'après un temps prolongé.

f) des accélérations prolongées de 6°/sec sec et 12°/sec sec pendant lesquelles l'activité subit les modifications déjà décrites.

g) enfin l'arrêt de la rotation à vitesse uniforme sous l'influence duquel l'activité subit une vive excitation suivie d'une phase inverse d'inhibition importante.

On peut constater au cours de cette expérience que la phénomène d'affaiblissement de la réponse est aussi manifeste pour un sens de rotation que pour l'autre. Aussi longtemps que la cupule reste défléchie l'activité qu'elle ait été excitée ou inhibée sous l'effet du stimulus pour un temps prolongé tente de retrouver son niveau de base. Si la cupule pendant l'évolution de ce phénomène est elle-même en voie de retour à sa position de repos l'activité dépasse ce niveau normal et se trouve pour un temps prolongé soit en dessous soit en dessus du niveau idéal qu'elle cherchait à atteindre.

Nous avons d'autre part mesuré le voltage de ces effets inverses lors d'arrêts brusques de mouvements rotatoires uniformes de vitesses variées. Ces effets inverses sont pour le même stimulus assez variables d'une préparation à l'autre. Parfois ils sont très marqués ou au contraire à peine décelables, prolongés (100-200 sec) ou relativement brefs (30-40 sec). Sur la même préparation nous avons constaté que les effets inverses diminuaient d'ampli-

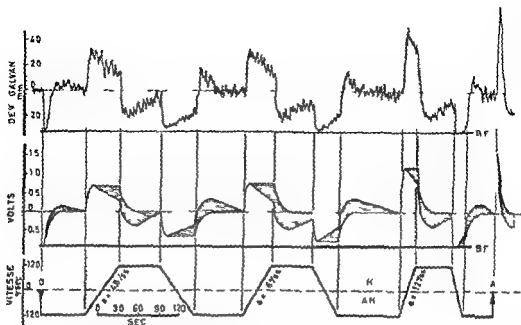


FIG. 4. 1. L'enregistrement au galvanomètre des variations de l'activité du nerf ampullaire droit d'une grenouille sous l'effet du démarrage de périodes de rotation à vitesse constante et d'accélération de durée prolongée et d'un arrêt brusque de la rotation (BF = bruit de fond des amplificateurs).

2. Traduction en volts des modifications (excitation et inhibition) de l'activité spontanée (0) de l'enregistrement 1. En hachuré le voltage qui sépare le niveau d'activité enregistré du niveau qu'il est possible de prévoir par la théorie du pendule de torsion.

3. Evolution de la vitesse angulaire pendant le déroulement de l'expérience (D = démarrage, A = arrêt, a = accélération, H = sens horaire, AH = sens antihoraire).

tude avec le vieillissement et disparaissent les premiers bien avant les réponses à la rotation et l'activité spontanée. Enfin si souvent les effets inverses sont de même amplitude de l'un et l'autre cotés de l'activité spontanée nous les trouvons dans certaines préparations très marqués en inhibition et faibles en excitation ou vice versa. En général cependant on constate que l'intensité et la durée de l'effet inverse augmentent progressivement avec le stimulus et l'on peut même préciser avec son logarithme (fig. 7).

2. Maintien expérimental de la cupule en une position défléchue

La compression du canal semi-circulaire externe membraneux détermine une déflexion de la cupule vers l'utricule, déflexion qui s'accompagne d'une vive excitation de l'activité spontanée. Si cette compression est incomplète la cupule regagne sa position d'équilibre initial mais la friction augmentant et l'endolymphe rencontrant un obstacle de plus en plus serré à son libre mouvement le temps mis pour atteindre cette position est de plus en plus long suivant que l'on exerce des pressions plus accentuées sur le canal membraneux. Quand l'obstacle est complet la cupule qui fonctionne comme une valve mobile mais hermétique dans l'impoule ne peut plus revenir à sa

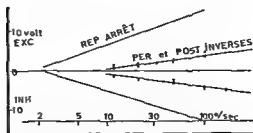


FIG. 5. Intensités comparées de l'effet inverse (per et post rotatoires) et des réponses primaires d'arrêt en fonction de la vitesse de rotation.

position d'équilibre. Elle reste par conséquent défléchie définitivement vers l'utricule.

Et cependant, comme on peut le voir sur la fig. 6 (C_4), l'activité revient progressivement mais très lentement (140 sec) à son niveau de base. Ce retour est très différent de celui qui suit les compressions non complètes du canal ou l'activité descend sous le niveau de base lors de retour de la cupule à sa position d'équilibre.

3. Déflexion permanente de la cupule par changement de position de la tête

Lors de l'étude que nous avons entreprise de l'activité électrique des canaux verticaux (Ledoux 1958) nous avons observé un phénomène non encore décrit à l'heure actuelle. Tout changement de position de la tête dans un plan vertical détermine, outre une brève excitation (mouvement liquidien ampullofuge) ou inhibition (mouvement ampullopete) de l'activité due au mouvement, une modification intense et prolongée de l'activité caractéristique de la position prise par le canal semi-circulaire.

La fig. 7 représente l'enregistrement de la réponse du canal vertical antérieur lors du maintien de la tête dans une position telle que le canal se trouve ampoule en bas (excitation) ou ampoule en haut (inhibition). Ces réponses bioélectriques s'affaiblissent progressivement pour atteindre après trois ou quatre minutes le niveau d'activité de base.

Nous avons interprété ces réponses présentes dans toutes nos observations comme résultant d'une prise de position nouvelle de la cupule. Il est probable en effet que celle-ci présente une densité différente de celle des liquides qui

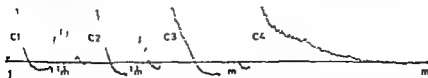


FIG. 7. Enregistrement au galvanomètre de l'effet sur les réponses du canal externe de cinq séries (C_1 à C_4) de plus en plus poussées du canal externe (C_4 = compression totale). m = mouvements de la tête permettant de contrôler les mouvements encore possible du système en l'absence de la cupule.

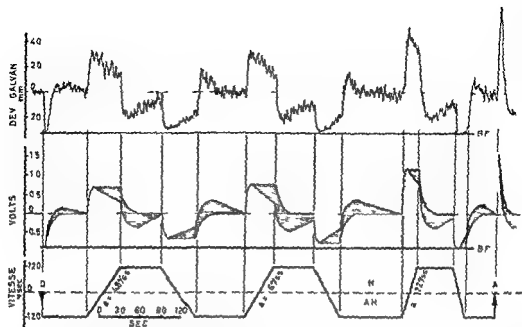


FIG. 4-1. L'enregistrement au galvanomètre des variations de l'activité du nerf ampullaire droit d'une grenouille sous l'effet du démarrage de périodes de rotation à vitesse constante d'accélérations de durée prolongée et d'un arrêt brusque de la rotation (BF = brusque de fond des amplificateurs)

2 Traduction en volts des modifications (excitation et inhibition) de l'activité spontanée (0) de l'enregistrement 1. En hachure le voltage qui sépare le niveau d'activité enregistré du niveau qu'il est possible de prévoir par la théorie du pendule de torsion

3 Evolution de la vitesse angulaire pendant le déroulement de l'expérience (D = démarrage A = arrêt a = accélération H = sens horaire A H = sens antihoraire)

tude avec le vieillissement et disparaissent les premiers bien avant les réponses à la rotation et l'activité spontanée. Enfin si souvent les effets inverses sont de même amplitude de l'un et l'autre côtés de l'activité spontanée nous les trouvons dans certaines préparations très marquées en inhibition et faibles en excitation ou vice versa. En général cependant on constate que l'intensité et la durée de l'effet inverse augmentent progressivement avec le stimulus et l'on peut même préciser avec son logarithme (fig. 5)

2. Maintien expérimental de la cupule en une position défléchie

La compression du canal semi-circulaire externe membraneux détermine une déflexion de la cupule vers l'utricule déflexion qui s'accompagne d'une vive excitation de l'activité spontanée. Si cette compression est incomplète, la cupule regagne sa position d'équilibre initial mais la friction augmentant et l'endolymphe rencontrant un obstacle de plus en plus serré à son libre mouvement le temps mis pour atteindre cette position est de plus en plus long suivant que l'on exerce des pressions plus accentuées sur le canal membraneux. Quand l'obstacle est complet la cupule qui fonctionne comme une valve mobile mais hermetique dans l'ampoule ne peut plus revenir à sa

trouvent diminuées pendant l'évolution du phénomène d'adaptation et ne donnent qu'une idée inexacte du mouvement réel de la cupule.¹

D'autre part enfin, la phase inverse connue depuis BARNY (1907) et Buys (1924) et observée aussi bien au cours des réponses rotatoires que des réponses caloriques, trouverait dans le phénomène d'adaptation, une explication satisfaisante, explication que ne nous a pas donnée jusqu'à présent, ce que nous savons du mouvement cupulaire lui-même.

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DISCUSSION

R. Mittermaier. Seitdem Buys den phasenhaften Wechsel der Vestibularrichtung unter bestimmten Versuchsbedingungen beobachtet hatte, hat man dieser klinischen Erscheinung viel Beachtung geschenkt. Herr Ledoux hat soeben einen wichtigen experimentellen Beitrag zur Erklärung dieses Phänomens gegeben.

¹ Si nous corrigions par exemple en tenant compte de la latence les valeurs données plus haut pour la grenouille nous trouvons pour $\pi \Delta$ 10 sec

$$\theta \Delta - 0.02 \quad \xi = 0.02 \alpha \left(1 + \frac{1}{e^{0.11}} \right)$$

et après un temps infini $\xi_{\infty} = 0.02 \alpha$

Die Elektroneurophysiologie kennt das sogenannte positive Nachpotential nach kurzzeitigem Spitzenpotential, das sehr deutlich in der Form der Summierung der Nachpotentiale nach einer frequenten Serie von Reizen in Erscheinung tritt (Gasser, H S, *The control of excitation in the nervous system* Harvey lect 32, 169 193, 1936/37)

Das periodisch gedämpfte Wiedereinschwingen nach einem kurzzeitigen Reiz und zwar diesmal als eine Erscheinung, die sich unter Umständen über Minuten erstreckt, ist bei vielen Reaktionen zu beobachten, denen eine Regulationsfunktion zu Grunde liegt. Zum Beispiel beim Pupillenreflex nach kurzdauerndem Lichtreiz oder bei dem Blutzuckergehalt nach stossartiger i.v. Traubenzuckerbelastung (siehe bei H. Drischel in *Regelungsvorgänge in der Biologie*, München 1956)

Das phasenhafte Auspendeln des experimentellen Nystagmus ist wohl von diesen Gesichtspunkten aus am besten zu verstehen.

Herr Ledoux erwähnt auch den Nystagmusablauf nach langdauernden Reizen. Ich habe vor mehreren Jahren einmal den Nystagmus während einer fast $\frac{1}{2}$ stündigen Kaltpulung registrieren lassen. In der ersten 2 Minuten war der Nystagmus sehr lebhaft, dann wurden die Amplituden kleiner, in den 11 Minute war er sehr unregelmässig, in der 8 Minute wurden die Schläge wieder deutlicher und regelmässiger, um von der 9 Minute an nur noch sehr unregelmässig in Erscheinung zu treten. Von der 13 Minute an war der Nystagmus nur noch hin und wieder zu erkennen. Ein solcher Versuch ist für die Versuchsperson wenig angenehm. Ich habe ihn deswegen nicht wiederholt. Bei der Registrierung ist auch mit Störungen zu rechnen. Ich glaube dass die Wiederholung eines solchen Versuches im Tierexperiment recht aufschlussreich bezüglich der Wirkung eines Dauerreizes sein sollte.

A. Ledoux (Réponse). Je remercie très vivement mon ami Hennebert ainsi que les Professeurs Mittermaier et Montandon qui ont bien voulu montrer un réel intérêt pour le problème posé dans cette communication.

Je puis préciser au Dr. Hennebert que l'activité électrique que nous avons décrite provient du nerf ampullaire coupé entre l'électrode et le système nerveux central. L'adaptation signalée ne peut dès lors être que périphérique.

Je sais aussi comme le Professeur Mittermaier le rappelait, que l'adaptation est un phénomène très connu en physiologie. Je crois d'autre part utile de rappeler qu'il est dangereux de parler de stimulation continue et uniforme du système cupule-endlabyrinthe dans le cas d'une épreuve calorique prolongée. Les mouvements de convection du liquide se modifient et s'épuisent avec le réchauffement ou le refroidissement progressif de tout le système. C'est d'ailleurs une constatation expérimentale que nous avons pu faire chez la grenouille.

Je dois enfin avouer mon embarras devant la question du Professeur Montandon dont l'immense expérience en nystagmographie m'oppose un argument de poids. Puis je cependant lui faire remarquer qu'il s'agit ici d'un fléchissement dans l'intensité de la réponse. Il n'y a rien qui concerne le nystagmus. Ce fléchissement d'intensité ne peut se mesurer comme on le sait à l'heure actuelle que par l'appréciation de la vitesse de la phase lente. Puis je demander à mon tour au Professeur Montandon s'il a étudié de façon systématique les variations de vitesse de la phase lente au cours de ses épreuves ? Le Professeur Jongkees me confirme en effet à l'instant, qu'il a observé et décrit à la réunion de Venne un ralentissement de la phase lente du nystagmus au cours d'accéléérations continues prolongées.

GAMMAGLOBULIN TREATMENT AND PROTECTION AGAINST INFECTIONS

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The effect of gammaglobulin treatment on the frequency of relapse of acute otitis in children can be summarized as follows: (1) the risk of a first relapse is on an average about 3% for untreated patients and about 1.5% per month for gammaglobulin treated patients; (2) both for untreated and for treated patients the risk of relapse is greatest about 2 months after the otitis, smallest about 6 months after the otitis, with another maximum about 12 months after the otitis; (3) the risk of a second relapse is on an average about 3% per month both for treated and for untreated patients; (4) relapses of third and higher order occurred in the untreated but not in the treated patients.

History

In 1951 the Swiss clinician Löffler described a case of recurrent pneumococcal meningitis in a 47-year-old man who had had 12 relapses in the course of half a year. In this patient a complete absence of gammaglobulin from the plasma was demonstrated. Since the gammaglobulin fraction in plasma includes the antibodies, the absence of this fraction would imply that at least one of the body's defence mechanisms against infections—the formation of antibodies—does not function. This was how Löffler explained the frequent recurrences in his case—the patient had also *keine Indikation für Abwehrmöglichkeit von Seiten des Organismus*.

Bruton *et al.* (1952) and Janeway *et al.* (1953) in particular have by their investigations shown that absence of gammaglobulin manifests itself clinically in frequent and very severe bacterial and virus infections, and that it appears in two forms. One form is a sex-linked recessive genetic defect which is present in half the number of boys born by women who carry the gene but are otherwise clinically normal with respect to infection. The other form is not inherited but acquired and occurs in both men and women at various ages. Nothing is known about its cause.

Surveys of pertinent problems and case reports have been published by, for instance, Kulneff, Pedersen & Waldenström (1955), Barrett & Volwiler (1957) and Kallös (1958). The complicated immunological problems are discussed in detail by these authors. Agammaglobulinaemia is said to be a manifestation of insufficiency in the lymphocytic and plasma cellular (antibody producing) system. This insufficiency is seldom total. Partial immunological insufficiency may be present, however, without any demon-

strable quantitative change of the plasmaglobulin level. Accordingly, those cases in which marked deficiency of gammaglobulin—hypogammaglobulinaemia—can be diagnosed are to some extent extreme cases. On the other hand, cases with marked hypergammaglobulinaemia as in myeloma and sarcoidosis are also extreme cases which from an immunobiological view point resemble the hypogammaglobulinaemic ones in that they too are defenceless against infections (Zinneman *et al.* 1954; Sones & Israel 1954).

It has been shown that the frequent infections in patients with an immunological defect can be prevented by regular administration of human gammaglobulin from healthy donors (pooled gammaglobulin). The optimal dose is 0.1 g of gammaglobulin per kg of body weight given intramuscularly once a month (0.1 g of gammaglobulin = approximately 0.9 ml of the 12% solution available on the market).

Harris & Schick (1953, 1954) were the first to suggest that even in persons with quantitatively normal values for plasmagammaglobulin there may be a complete or partial deficiency of antibody formation to certain antigens. This deficiency may influence the course of some infections. Harris & Schick describe 6 cases of respiratory tract and/or gastro-intestinal infections in children which were resistant to antibiotics and ran an unusually severe and prolonged course or showed a tendency to frequent relapses. According to the authors, administration of human gammaglobulin led to definite improvement in these cases. Knouf (1957) describes ten cases of resistant infection in which gammaglobulin was of obvious benefit. Schonholtz *et al.* (1958, 1959) present a more homogeneous material and demonstrate the potentialing effect of gammaglobulin on antibiotic therapy. Their series consisted of 19 patients with severe osteomyelitis who received chloramphenicol and gammaglobulin (17 cases) and novobiocin and gammaglobulin (two cases). The first named combination had a good effect in 12 patients. At least ten of these had earlier been treated with chloramphenicol alone for a prolonged period and in large doses without a definitive effect. In ten out of all the cases the result of the combined treatment was designated as excellent in two cases as good and in two as fair. Barandun *et al.* (1959) have recently published a detailed report of 66 cases of extremely severe therapy-resistant bacterial infections in normogammaglobulinaemic patients. In at least 31 of these cases administration of gammaglobulin led to an unmistakable change for the better of the clinical picture. In the other 35 cases, konnte eine mehr oder weniger ausgeprägte therapeutische Wirkung des Gammaglobulins nicht ausgeschlossen werden. The authors state that in ten cases the therapeutic effect of the gammaglobulin was bei kritischer Bewertung aller verfügbaren klinischen Daten the only possible explanation of the very obvious improvement. In the rest of the cases the possibility that other therapeutic measures too may have played some part cannot be excluded. Stream *et al.* (1958) report encouraging results of treatment of recurring oral herpetic lesions with gammaglobulin alone in relatively large doses.

All the authors quoted above seem to have presumed that the administered

human gammaglobulin contained specific antibodies. This was not demonstrated by any tests, however.

Loughlin *et al* (1958-1959) obtained good results in 11 cases of severe secondary and ten cases of severe tertiary lymphogranuloma venereum by combined gammaglobulin and chloramphenicol therapy. They state that this combination is definitely more effective than chloramphenicol alone or for that matter any antibiotic administered singly in the treatment of advanced secondary or tertiary lesions of lymphogranuloma venereum. In their opinion it is however highly improbable that the human gammaglobulin used would have contained specific antibodies to the virus of lymphogranuloma venereum and so there must be some other explanation of the beneficial effect of the gammaglobulin in these cases.

The above mentioned clinical results of treatment are to some extent supported by various experimental studies in animals. It has for instance under standardized conditions been conclusively shown that human gammaglobulin potentiates the effect of chloramphenicol in mice infected with *S typhimurium*, *E coli* and *D pneumoniae* (Manning *et al* 1957, 1958). Huck & Hiser (1958, 1959) have confirmed these results and moreover found that gammaglobulin also potentiates the effect of tetracyclines under similar conditions and that a corresponding potentiation can also be demonstrated in cases of infection by *Staph aureus* (Smith's strain). Roodey *et al* (1957, 1958) were able to show that in mice infected with *Strep pyogenes* subcurative doses of chloramphenicol or penicillin combined with subcurative doses of gammaglobulin resulted in a high survival rate. Holper (1957-58) has shown that human gammaglobulin potentiates the effect of ristocetin in mice infected with *Staph aureus* or *Strep pyogenes*.

An experimental investigation that greatly contributes to our knowledge of the efficacy of human gammaglobulin in protecting against bacterial infection has recently been reported by Zimmermann (1960). In comprehensive serial investigations on mice he was able to show that human gammaglobulin in 16% solution (1 ml per animal of which 0.5 ml was injected intraperitoneally and 0.5 ml intravenously 18 hours before and 0.25 ml intravenously two hours after the experimental infection) definitely conferred protection against an infection by toxic *L coli* strains, *Streptococci* or *Staphylococci* which caused a fatal result in control mice within 24 hours and against a mixed infection by *F coli* and *Streptococci*. The combination of gammaglobulin and sublethal doses of sulphonamides or certain antibiotics (such as streptomycin or tetracyclines) gave such unequivocal results that the author considers this method to be die derzeitig wirksamste Behandlungsmethode bei allen nicht genau erreg器definierten bzw. schweren Allgemeininfektionen.

This survey of the literature shows that the effect of gammaglobulin treatment in animals has been established whereas in man the results are less convincing partly because trials have been made in only a small number of cases, controls are lacking and the course of the disease has in some cases not been very clearly defined.

Own Investigations

In dealing with upper respiratory-tract infections it is often difficult to define clearly one particular disease. Our investigation into the prophylactic effect of gammaglobulin treatment is based on a series of children with acute otitis. Such cases are clearly defined and distinguished from other clinical syndromes and are thus well suited for a study of this kind. The division into a "treated" and a "control" group was based on the date of birth. Patients born on an odd date were given gammaglobulin and those born on an even date were not given gammaglobulin.

Not considering the gammaglobulin, the treatment was for both groups as follows. Paracentesis was done as early as possible, followed by washing out of the ear and instillation of spir. acid. boric (when there was risk of permanent perforation of the ear drum, insufflation of boric acid powder was used instead). The treatment was carried out either in the patient's home or in the ear department, according to the nature of the case and the social circumstances. If the disease lasted a week without conclusive evidence of healing, that is, absence of fever, decrease or cessation of aural discharge, a drum clearing up and improvement of hearing and, of course, absence of any signs of complications, antibiotic therapy was instituted.

The assessment of the therapeutic effect in cases of acute otitis offers great difficulties, however. Only the initial symptoms are distinct. They are earache, fever, impaired hearing, and aural discharge starting relatively early. The course of acute otitis up to healing, on the other hand, varies considerably and is not easily assessed. This is what makes the recording of the therapeutic effect difficult. To avoid the difficulties of assessing the effect of gammaglobulin in our investigated series we did not base our comparisons on the course of the acute otitis. We used instead the frequency of relapse of acute otitis.

From earlier investigations it is known that the incidence of otitis in children is five to ten times higher than that in adults. By including only cases of acute otitis in children we could expect to collect, within a reasonable period of time, a large number of cases, among which a large number of relapses could likewise be expected within a reasonable period of time. The investigation was started in February 1958 and was continued for about 1½ years. It comprises all the cases of acute otitis in children between 1 and 7 years of age, who during the period of investigation attended the Ear, Nose and Throat Department in Halmstad. All the children born on an odd date were given gammaglobulin at their first visit and then once a month for half a year. The children born on an even date were not given gammaglobulin. In other respect the two groups received the same treatment, as outlined above. Gammaglobulin¹ was administered in a dose of 0.6 ml/kg of 12% solution, which is a slightly lower dose than that used in agammaglobulin.

¹ Gammaglobulin was placed at our disposal by Åli Kabi, Stockholm.

aemia. In our series all the patients had values for gammaglobulin that were normal for their ages.

In recording the first relapse we did not calculate in calendar months but took the first gammaglobulin treatment as month 0. All the patients during 8 consecutive calendar months were included in the series. The main purpose of the investigation was to compare the two groups and since these run parallel there will be no seasonal influence on this comparison. The patients in the 'treated' group attended the clinic once a month to receive a dose of gammaglobulin. This ensured direct checking of the frequency of relapses. As regards the control group the parents of the children were given stamped and addressed post cards which they returned once a month with information about the children's state of health. In this way a check was kept on these children too. (The same post card system was also used for the 'treated' group as a means of continued checking after termination of 6-7 months gammaglobulin treatment.)

RESULTS

The 'treated' group consisted of 113 and the control group of 118 children. The above described principle of selecting the cases assured a random selection and a check up showed that the two groups could be regarded as homogeneous with respect to age, sex, etc. For various reasons 34 children did not continue the prescribed treatment through 6-7 months. This means that only 79 were fully treated. Since discontinued treatment would reasonably produce an effect midway between no treatment and a full course treatment the result ought to have been a lower average therapeutic effect. Accordingly the assessment that will be made of the effect of gammaglobulin treatment will underestimate the effect.

Another deviation from the original plan was that a total of six patients in the untreated group received gammaglobulin after their first relapse. Obviously this does not influence the result as regards the occurrence of first relapses but it may influence the result for second and third relapses so that the difference between treated and untreated cases may be smaller than it would have been if we had followed the original plan. This too should lead to underestimation of the therapeutic results.

The number of first relapses in the different months for the treated group will be seen in Fig. 1 and that for the untreated control group in Fig. 2. Fig. 3 shows the total number of children (treated and untreated) who had a first relapse. The black bars represent first relapses that during the period of observation were followed by second relapses. It will be seen that only first relapses occurring in the beginning of the period of observation were followed by second relapses.

The frequency of relapses seems to be unevenly distributed over the different months following the original attack. Statistical analysis showed that there were significant differences between the different means. According to a

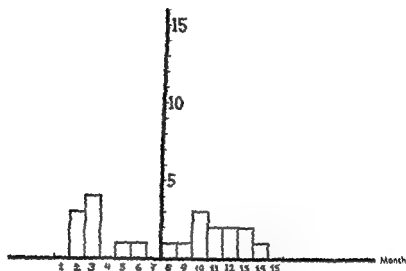


Fig 1 No of 1st relapses, treated group

preliminary study these differences are not related to calendar months but seem to suggest some correlation with the time from the first otitis to relapse.

Fig 4 illustrates the risk of first relapses as a percentage per month for the treated and the untreated group. In order to eliminate some of the random month-to-month variations, five month means have been plotted in the diagram, that is, a point represents the mean for those five months that lie around the particular point. The first and the last point on each curve represent analogously calculated means for three months. This method does not permit a study of short variations in the risk of relapse but only of the course as a whole. It will be seen that the risk of relapse for the treated cases is throughout about half that for the untreated control cases. For both groups

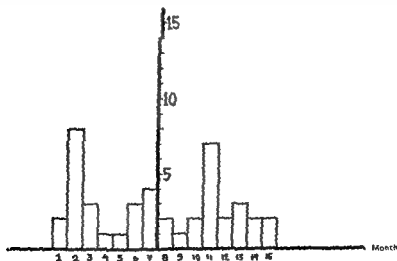


Fig 2 No of 1st relapses, untreated group

TABLE 1 Risk of relapse per month as a mean for the whole period of time

	1st relapse %	2nd relapse %	3rd relapse %
Treated cases	14	30	None
Untreated cases	27	34	11.8

the risk is great around the second month falls to a minimum around the sixth month and reaches another maximum about the 12th month after the otitis

The risk of second relapse has been calculated for those who have already had a first relapse and the risk of a third relapse for those who have already had a second relapse

The treatment seems to influence notably the first relapses. The risk has fallen to half the corresponding figure for the untreated cases. The risk of second relapse seems to be largely unchanged. In the treated group there are only first and second relapses in the untreated one there are relapses of third and higher order. Accordingly two patients had three relapses and two patients five relapses each during the period of observation. Consequently, the treatment seems to have protected against relapse of higher order.

To find out which effects of gammaglobulin treatment are statistically significant the number of first and second relapses was calculated in different periods of time from month 1 to month 15 and the number of third relapses from month 1 to month 15 both for treated and for untreated cases.

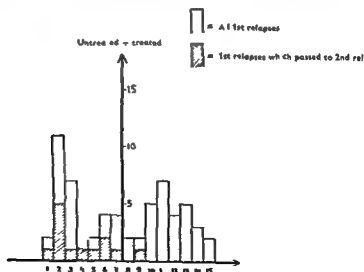


Fig. 3 % of 1st relapses all cases

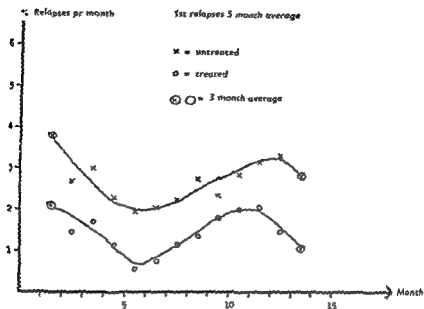


Fig 4 Risk at 1st relapse

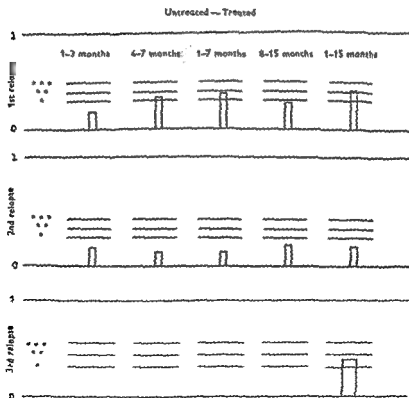


Fig 5 Differences in t values shown in stem diagrams. The difference must exceed for * ≥ 2.8 , for ** 0.340 and for *** 0.133

TABLE 2

			1st-3rd month	4th-7th month	1st-7th month	8th 15th month	1st 15th month
Treated patients (113 cases)	1st relapse	No. of relapses		2	11	12	21
		% relapses	6.20	1.7	7.96	10.67	18.58
		χ^2	0.509	0.767	0.571	0.664	0.891
	2nd relapse	No. of relapses	0	1	1	3	4
		% relapses	0	0.88	0.88	2.66	3.54
		χ^2	0.091	0.185	0.188	0.328	0.378
	3rd relapse	No. of relapses	—	—	—	—	0
		% relapses	—	—	—	—	0
		χ^2	—	—	—	—	0.001
Untreated patients (118 cases)	1st relapse	No. of relapses	13	9	22	18	40
		% relapses	11.01	7.62	18.63	15.25	33.88
		χ^2	0.676	0.559	0.892	0.802	1.242
	2nd relapse	No. of relapses	7	3	3	8	0
		% relapses	5.93	2.54	2.54	6.78	0
		χ^2	0.261	0.320	0.370	0.527	0.550
	3rd relapse	No. of relapses	—	—	—	—	4
		% relapses	—	—	—	—	3.38
		χ^2	—	—	—	—	0.300

The degree of significance was determined by the use of the function $\chi^2 = 2n \ln \sin \frac{1}{2}p$ which is normally distributed with the variance $1/n$ independent of the value of p and hence of χ^2 . p is here the fraction of the original group that had for instance a first relapse and n is the number of cases in the original group. If $p = 0$ Bartlett's correction is used which gives $\chi^2 = 2n \ln \sin \frac{1}{4} \sqrt{n}$.

Table 2 records the number of relapses over the different periods, the corresponding percentages of the original group and the corresponding value of χ^2 . Table 3 shows the differences in the χ^2 value between the untreated and the treated group and corresponding significances. The same values are

TABLE 3 Differences in χ^2 values

	1st-3rd month	4th-7th month	1st-7th month	8th 15th month	1st 15th month
1st relapse	0.173	0.712*	0.321*	0.238	0.351**
2nd relapse	0.167	0.132	0.132	0.19	0.181
3rd relapse	—	—	—	—	0.27*

shown graphically in Fig 3. The horizontal lines indicate the significance levels *, **, ***. All the comparisons show that the treated patients had fewer relapses than the untreated ones. As regards the first relapse a significant difference was obtained for the period from the 4th to the 7th months inclusive (*) for the period from the 1st to the 7th months inclusive (*) and for the whole period (1st to 10th months) (**). The comparison concerning the whole period must here be considered to give the most relevant measure of the effect of the gammaglobulin treatment as the risk of relapse for treated and untreated cases goes approximately parallel during the whole observation period (see Fig 4). The difference in the number of third relapses (4 in the untreated 0 in the treated cases) is significant for the whole period (*).

A fact of greatest practical importance seems to us to be that the frequency of relapse in the treated group for the whole period of observation (10 months) was only about half of the corresponding frequency in the untreated group. This difference is statistically significant (**). Moreover in the untreated group some patients had up to five relapses whereas in the treated group no patient had more than two relapses. This difference too is statistically significant (*).

ZUSAMMENFASSUNG

Die Wirkung der Behandlung mit Gammaglobulin kann wie folgt zusammengefasst werden: (1) das Risiko für einen ersten Rückfall beträgt per Monat bei unbehandelten Patienten ca 3% und bei mit Gammaglobulin behandelten Patienten ca 1.5% (statistisch sichergestellte Differenz); (2) in beiden Gruppen ist das Risiko für ein Rezidiv am grössten ca 2 Monate nach der Otitis am geringsten ca 6 Monate nach der Otitis mit einem zweiten Maximum ca 12 Monate nach der Otitis; (3) das monatliche Risiko für ein zweites Rezidiv beträgt sowohl für behandelte als auch für unbehandelte Fälle etwa 3%; (4) dritte und mehrmalige Rezidive traten ausschliesslich in der unbehandelten Gruppe auf somit nicht bei mit Gammaglobulin behandelten Patienten.

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APPROACH TO THE GENERATION CENTRE OF NYSTAGMUS ALTERNANS BY MEANS OF DRUG TESTS¹

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Barbiturates and chlorpromazine stop nystagmus alternans temporarily. During the time of action of the barbiturate nystagmus can be elicited in the patient by caloric stimulation, whereas during the action of chlorpromazine there is no caloric reaction. From these observations the conclusion can be drawn, that the generation centre of the nystagmus alternans is above the primary vestibular reflex arch.

In 1958 two cases of nystagmus alternans were described by Dr. Károly Ozsváth and myself. We were intrigued by the mechanism of this rare and interesting pathological manifestation. We accepted in our paper the supposition of Ohm, Boenninghaus and others, according to which nystagmus alternans is the consequence of the disorder of the higher vestibular centres.

In the present study, on the basis of a new case, I have endeavoured to approach the generation centre of nystagmus alternans by means of laryctile and intranarcon.

In 1959 I made investigations upon the effect exerted by chlorpromazine on the vestibular system of healthy young men. I administered 5–10 mg of chlorpromazine and then, after the effect had set in, I stimulated the labyrinth with cold water. I found that chlorpromazine, even in such a small dose as 5–10 mg considerably diminishes or even altogether stops caloric nystagmus.

Fig. 1 displays two particular aspects of a nystagmogram. The first shows the maximum of the nystagmus manifested after caloric stimulation of the left ear of a healthy person by means of 150 cc of water at 20°C. The second shows that 18 minutes after the intravenous administration of 5 mg of laryctile the same caloric stimulation elicited a nystagmus of considerably smaller amplitude.

I investigated the effect of chlorpromazine on 10 healthy persons altogether and all of them showed a caloric nystagmus of considerably diminished intensity. In case of intravenous administration of 10 mg of laryctile, nystagmus was not elicited even with a massive stimulus.

¹ On the basis of a paper read at the Hugyes memorial session of the Hungarian Ophthalmoneurological Section on the 5th of October 1960.

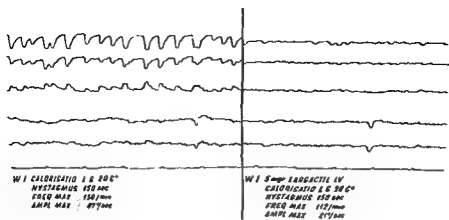


FIG 1

We know that, by stimulating the vestibular end organ, nystagmus may occur in two ways. One is via the fasciculus longitudinalis posterior, the other via the substantia reticularis. The experiment of Lorente de No is well known, in which he severed the fasciculus longitudinalis posterior and was yet able to elicit nystagmus, which is possible only if the substantia reticularis takes part as well in the conduction of the stimulus.

According to the literature, chlorpromazine affects in the first place the substantia reticularis. Our observation, that a small dose of chlorpromazine is able to stop caloric nystagmus entirely, proves that it paralyses not only the substantia reticularis, but the primary vestibular reflex arc as well.

The barbiturates, as is well known, affect the cortex and the subcortex. Blomberg in 1936, in his experiments made for other purposes, was able to produce caloric nystagmus during the action of barbiturate derivatives. Bender *et al* in 1946 found in the course of their evipan experiments that barbiturate derivatives also check the production of optokinetic nystagmus. These phenomena are to be explained with the fact that barbiturates affect in the first place the higher centres and do not check the primary vestibular reflex arc function.

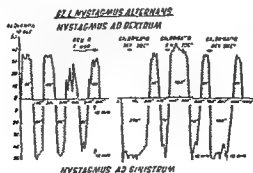


FIG 2.

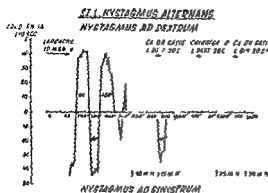


FIG 3

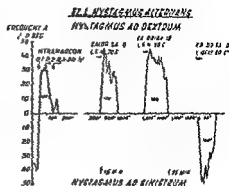


FIG 4

I endeavoured to utilize the difference between the effect of chlorpromazine and that of the barbiturates in the approach to the generation centre of nystagmus alternans as follows

Sz L, a 38 year old male, reported that his eyes had vibrated from childhood. He was in our neurological department in the autumn of 1959, when chronic neurosis was diagnosed. According to the ophthalmological examination, vision on both sides was 0.8. On the back poles of both lenses there were tiny opacities (cataracta polaris posterior). The patient can otherwise see well enough to be an excellent marksman.

The electronystagmographical data taken from the patient are plotted in Fig 2. On the abscissa of the graph are plotted the seconds, on the ordinate the number of strokes of the nystagmus in 10 seconds. The data of the nystagmus beating to the right are drawn above the line, those of nystagmus to the left below. It is easy to observe in the illustration that on looking straight ahead the nystagmus strikes to the right on the average for 100 sec, then turning shows its quick component to the left for 90 sec or so. These right and left phases of the nystagmus alternate with considerable regularity. External stimuli, such as the closing of the eyes or some caloric stimulus, could influence the rhythm of the turn of the nystagmus only moderately.

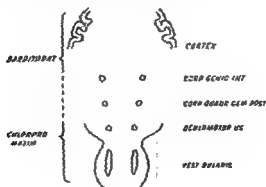


FIG 5

Thus this case was classified in the dominant (*reizwiderstandsfähiger*) group

I administered to the patient 40 cg of intranarcon intravenously. Intranarcon is a Hungarian product (allyl cyclohexenyl thiobarbituracide sodium).

On the action of the intranarcon the nystagmus alternans stopped and did not return during the 30 minutes of the examination. At the same time caloric reaction can be elicited from the 15th minute of the experiment onward. The duration and frequency of the nystagmus thus elicited is normal but the amplitude is considerably smaller than the normal.

At the next examination after a few days I gave 10 mg of largactil intravenously. The largactil stopped the nystagmus alternans in 10 minutes or so. In the 15th minute I succeeded in eliciting a rudimentary caloric nystagmus but from that time onward during the 30 minutes of the experiment, the eliciting of the caloric nystagmus was not possible.

From the experiment above described the following conclusion can be drawn. The generation centre of nystagmus alternans is above the primary reflex arch. This is proved by the fact that chlorpromazin which paralyzes the primary reflex arch and the substantia reticularis alike stops nystagmus alternans as well as caloric nystagmus whereas intranarcon acting in small doses cortically and subcortically stops nystagmus alternans but the caloric nystagmus can be elicited for the function of the primary reflex arch prevails. Thus the generative cause of nystagmus alternans can be only above the primary reflex arch.

ZUSAMMENFASSUNG

Barbituraten und Chlorpromazin beheben nystagmus alternans einstweilig. Während der Wirkungszeit der Barbituraten kann man beim Patienten mit kalorischer Stimulation nystagmus hervorrufen. Demgegenüber gibt es während der Wirkung von Chlorpromazin keine kalorische Reaktion. Von diesen Beobachtungen kann die Folgerung gezogen werden, dass das Entstehungszentrum von nystagmus alternans über dem primären vestibulären Reflexbogen ist.

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STUDIES ON HABITUATION OF VESTIBULAR REFLEXES

I *Effect of Repetitive Caloric Test*¹

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The phenomenon of habituation to repetitive caloric stimulation was investigated in cats. The study included acquisition, retention and transfer of the response decline in nystagmic reaction.

Acquisition of habituation was obtained by repetitive caloric stimulation of right and/or left ears with hot and/or cold water. The rate of response decline was rapid with the initial irrigations and then tended to level off. The extent of response decline differed among animals, ranging from moderate reduction to complete extinction.

In the cat, habituation may be retained after three weeks of rest; however, in several animals, recovery of normal caloric responses occurred after a few days.

Transfer of habituation occurs, provided the nystagmus is provoked in the same direction as that which was habituated.

The evidence supports the opinion that the neural mechanism underlying habituation of vestibular reflexes is a central process. The locus of this mechanism remains unknown.

The practical and theoretical implications of this phenomenon are briefly discussed.

The term "habituation" introduced by Abels (1906), is here used to indicate the phenomenon of progressive reduction of the nystagmic response (response decline) to either repetitive rotary or caloric tests.

Habituation to rotary tests has been observed in both man (Abels, 1906; Dodge, 1923; Dunlap & Dorcus, 1926; Griffith, 1920 and 1921; Hinkle, 1923; Wendt, 1931) and animals (Dettefsen, 1923; Fearing, 1937; Gould, 1926; Griffith, 1920; Halstead, 1935; Halstead, Yacorzynski & Fearing, 1937; Hood & Pfaltz, 1954; Maxwell, Burke & Reston, 1922; Mower, 1934; Some of these investigations (Mower, 1934; Halstead, 1935; Yacorzynski & Fearing, 1937) emphasized three characteristics of the phenomenon: acquisition, retention, and transfer. Acquisition, in this situation, stands for the progressive decrement of the nystagmic response.

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The rate of response decline is usually accelerated with the initial stimuli and then tends to level off. The extent may range from a moderate response decline to extinction; this depends on the conditions under which the rotary tests are presented. Retention seems to be the most important factor; no acquisition could occur if there were no persistence of the neural process underlying the response decline. Retention, however, is transitory in both man (McCabe 1960) and animals (Fearing 1941; Halstead 1935; Mowrer 1944). A few weeks of rest from rotary stimulation results in recovery of normal responses, but re-habituation can be rapidly established again. Transfer (Abels 1906; Dodge 1923; Halstead 1935; McCabe 1960; Mowrer 1934) means that the habituation to rotary stimuli in one direction produces equal or near equal response decline of vestibular responses to rotation toward the opposite direction.

It has been frequently postulated that habituation is a central nervous system phenomenon. The experimental evidence suggests strongly that the process is partially associated with a functional change in the cerebellum (Halstead 1935; Halstead, Macorzynski & Fearing 1937).

A few experiments carried out on habituation to repetitive caloric tests have given contradictory results. In man, response decline has been detected in the maximum velocity of the slow component (Hamersma 1957) but not in duration or frequency of the nystagmus (Hood & Pfaltz 1954). In animals, Dunlap (1925) produced habituation in rabbits, while Hood & Pfaltz (1954), working with the same species, did not. Henriksson *et al.* (1960) emphasized that the maximum velocity of the slow component in the cat diminished in magnitude within a few caloric tests repeated at short intervals.

The experiments to be reported here were undertaken for one purpose, namely, to investigate whether habituation to repetitive caloric tests in the cat is qualitatively similar to that established by rotary test in man and animals.

METHOD

The experiments were carried out in 61 adult healthy cats not exposed previously to caloric or rotary stimulation.

The techniques for immobilizing the cat, presenting caloric tests, recording nystagmus and evaluating the results have been described elsewhere (Henriksson, Fernandez & Kohut 1960). Unless otherwise specified, the caloric tests were done in full light in conscious animals.

The cats were fixed and oriented in space so that the horizontal canals were always in the vertical plane. Under these conditions, irrigation with water at 18°C or higher provoked horizontal nystagmus toward the irrigated ear, while with water at 28°C or lower, the nystagmus was toward the opposite side. It would be mentioned that irrigation of one ear with hot water or the opposite ear with cold provokes nystagmus in the same direction.

Irrigations of 40 seconds duration were done repetitively in one or both ears with water at either 0° 10° 20° 28° or 48°C. It is assumed that the stimulus strength at 48°C is equal to that at 28°C and that it increases as the temperature of the water is further decreased.

The results were evaluated in terms of both maximum velocity of the slow component and duration of the nystagmus. No other vestibular reflexes associated with caloric stimulation were utilized in this investigation.

Since several conditions of caloric stimulation were employed the data were organized with respect to a set of definitions adopted for convenience of exposition.

The term *equivalent stimuli* is applied to all caloric stimuli which regardless of their physical differences provoked a nystagmic response in the same direction. For instance water at 48°C applied in the right ear is considered equivalent to water at 28°C or less applied in the left. These stimuli provoked nystagmus toward the right. The responses produced by equivalent stimuli are called *unidirectional* which can be either toward the right or toward the left.

Stimuli which provoked nystagmus in opposite directions are called *nonequivalent stimuli* regardless of their physical identity. Water at 48°C for example applied in the right ear is not equivalent to water at 48°C applied in the left; the former provokes nystagmus toward the right, the latter toward the left. The responses elicited by nonequivalent stimuli are called *bidirectional*.

The term *hot water* is used when the temperature was 48°C; *cold water* is employed when the temperature was 28°C or lower.

Unless otherwise specified the term *trial* means a sequence of ten irrigations. The interval between any two successive irrigations was five minutes.

Eye velocity is a term chosen to represent the maximum velocity of the slow component.

When investigating the effect of trials (either retention or transfer of habituation) the term *test* is used. A test usually consisted of one irrigation, but in those cases where the outcome needed confirmation two irrigations were performed.

The terms *acquisition*, *retention* and *transfer* will be used for describing the main characteristics of habituation. The meaning of these terms has been already described; however the term *transfer* requires further explanation. Let us suppose that the right ear received one trial with water at 20°C and that the nystagmic response diminishes as the irrigations are repeated. Now we want to know whether the response of the same ear provoked by one irrigation with water at 48°C presents any change. If the change consists of a diminution of either the eye velocity or duration of the nystagmus then this phenomenon is called *transfer of habituation*. The testing for transfer should also be done in the opposite ear with either hot or cold water; the results are again evaluated in the manner explained above.

RESULTS

A Acquisition of Habituation

Among the numerous conditions which may modify the nystagmic response to repetitive caloric stimulation the following were investigated: stimulus strength, effect of both equivalent and nonequivalent stimuli, and anesthesia.

1 Effect of stimulus strength

In this series, each animal received one trial at either 0°, 10°, 20°, 28° or 48°C.

The observations consistently showed a response decline of eye velocity as the irrigations were repeated (Figs. 1 and 2). The duration of nystagmus was usually diminished. In some animals the magnitude of reduction was small and in a few there was no reduction.

The pattern of response decline was qualitatively the same for all temperatures. The eye velocity diminished rapidly with initial irrigations and then tended to level off. Both rate and extent of response decline, however, varied considerably among animals and certainly this variation was not associated with the stimulus strength.

Commentary. The importance of these experiments is that the phenomenon of habituation induced by repetitive caloric tests can also be demonstrated in the cat. The findings confirm previous observations made by Dunlap (1925) and Henriksson *et al.* (1960).

The pattern of acquisition as judged by both rate and extent of response decline follows closely that demonstrated by rotary tests in man and animals. This is not surprising because in either case (rotation and calorization) the stimulating mechanism of the receptor is the deflection of the cupula. The same neural process probably underlies acquisition of habituation to both repetitive rotary and caloric tests.

Two inferences of practical importance can be drawn from these observations. One: the caloric test in the clinic when repeated at short intervals should be interpreted cautiously. Two: the maximum velocity of the slow component seems to be a more sensitive index of vestibular function rather than duration of nystagmus.

Because of the theoretical and practical importance of the response decline to repetitive caloric stimulation, it would be desirable to conduct a thorough reinvestigation of this phenomenon in man.

2 Effect of equivalent stimuli

In this series two questions were investigated. First, whether acquisition of habituation is facilitated when equivalent stimuli are applied alternately in both ears. Second, whether transfer of habituation occurs.

The animals received one trial in which irrigation of one ear with cold water was alternated with irrigation of the other with hot water. The stimuli were equivalent and consequently the nystagmus was unidirectional. After

Habituation of Vestibular Reflexes

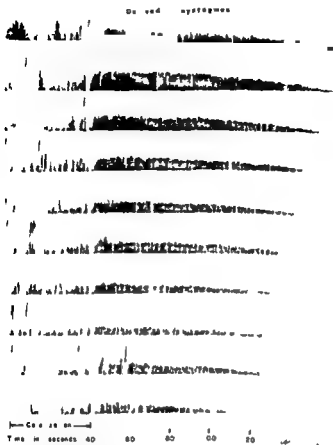


Fig. 1 Acquisition of habituation. Cat A 891 received one trial of ear. The large tracings which appear during calorization are negative, the cat is bent. Notice in this case a pronounced decline of habituation nystagmus.

completing the sequence of ten irrigations each day for transfer of habituation. This test consisted of the ear previously irrigated with cold water and reversed the direction of the nystagmus.

The observations revealed the same general pattern described in the previous series, but the response direction and it occurred at a faster rate. Reversing the direction, responses that were essentially normal.

Fig. 3 illustrates one case in which irrigations right ear alternated with 48°C in the left. An eye irrigation was confirmed by nystagmography and responses were obtained when the order of irrigation.

Commentary. These observations suggest that to alternate ears of the cat facilitate acquisition

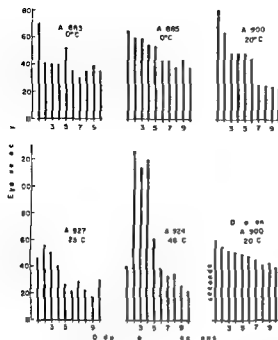


FIG 2

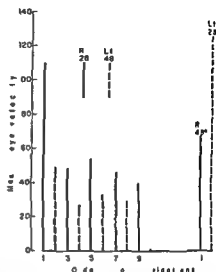


FIG 3

FIG 2 Acquisition of habituation as a function of stimulus strength. These representative cases illustrate the finding that the rate and extent of response decline of the eye velocity is independent of the stimulus strength. Duration of the nystagmus also diminishes as shown for cat A 900 but both the rate and extent of this diminution are moderate. To find duration of nystagmus for cat A 900 multiply ordinate by a factor of 4.

FIG 3 Effect of equivalent stimuli. Cat A 930 received one trial consisting of irrigations with water at 28°C in the right ear alternating with water at 48°C in the left ear. The direction of nystagmus was always toward the left. The tenth irrigation produced no response. Reversing the direction of nystagmus revealed a normal reaction indicating no transfer of habituation to non equivalent stimuli. In this and following figures calorizations of right and left ears are indicated by solid and broken bars respectively.

surprising effect that complete extinction may be obtained. These findings confirm those of Dunlap (1925) in the rabbit.

In the experiments on habituation to repetitive rotary tests the phenomenon of transfer has been observed (Halsted 1935 Halsted & Moravcsik & Fearing 1937 Mowrer 1934). It is implicit in the writings of these investigators that the same process which is responsible for the response decline of nystagmus following repetitive rotary tests in one direction is operating when nystagmus is provoked by rotation in the opposite direction. This type of interpretation is not supported by our results on caloric stimulation. It can be stated that habituation of an unidirectional nystagmus has no or little effect upon the nystagmus provoked in the opposite direction when this is tested for transfer. The disagreement between this statement and that based upon experiments with rotary stimulation is only apparent and will be discussed later.

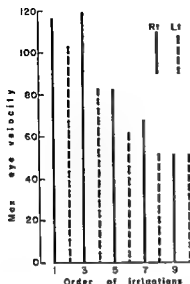


FIG 4

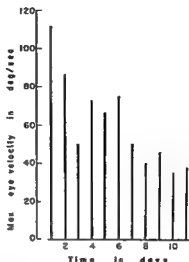


FIG 5

FIG 4 Acquisition of habituation to nonequivalent stimuli Cat V 941 was irrigated ten times with water at 20°C applied alternately to right and left ears. The provoked nystagmus was bidirectional and both showed response decline.

FIG 5 Retention of habituation Cat V 923. This animal received one trial of eleven irrigations each applied once a day in the right ear with water at 20°C. The pattern of response decline was similar to that obtained with irrigations repeated at five minute intervals.

3 Effect of nonequivalent stimuli

In this series habituation of bidirectional nystagmus was investigated. Two procedures were used: one, alternate irrigation of one ear with cold and hot water; the other, alternate irrigation of both ears with water at the same temperature.

The two procedures produced habituation of the bidirectional nystagmus, but the second procedure appeared to be more efficient. The rate and extent of response decline followed the pattern described in the previous chapter. An example illustrating these findings is presented in Fig 4.

Commentary. In this series the nystagmus was provoked alternately toward the right and left. The results showed that both responses, i.e. the nystagmus toward the right and left, were diminished in the same fashion. The pattern of response decline elicited from each ear was not different from that expected by a single trial of comparable temperature in that ear. This suggests that habituation of the nystagmic response in one direction has little or no effect upon habituation of nystagmus in the opposite direction.

4 Effect of anesthesia

Animals under pentobarbital sodium (0.7 cc per kg body weight) received one or more trials with water at 20° or 10°C in one ear. No nystagmo-

graphic recordings were taken during these trials simply because nystagmus is not present when the animal is under anesthesia. After recovery from the anesthetic (48 to 72 hours) the animals were tested to find out whether habituation had occurred.

The observations showed consistently that the nystagmic responses after recovery were normal. Some of these animals were given another trial under normal conditions and the characteristic response decline was clearly apparent.

A few animals under ether anesthesia also received one trial in one ear with water at 20°C. Approximately four to five hours later the cats were tested with water at 20° and 48°C in both ears. The results indicated that the nystagmic response toward both the right and left were depressed.

Commentary. The findings suggest that no habituation occurs under habiturate anesthesia. Since testing was done two or three days after the irrigation it may be argued that in the interim recovery had occurred. The results of trials following ether anesthesia suggest that habituation can take place in the anesthetized state. However, in this situation the response decline may have been due to the prolonged effect of the anesthetic. These data are difficult to interpret because factors other than caloric stimulation may have been influencing the tests.

The question whether acquisition takes place under general anesthesia is indeed important for the understanding of the neural process involved in habituation. A more thorough investigation should be carried out which would incorporate more stringent controls concerning retention of habituation under general anesthesia.

B Retention of Habituation

The concept of retention implies that one caloric stimulation produces changes in the vestibulo-ocular reflex arc which persist and accumulate with those produced by subsequent stimuli.

This interpretation gives rise to two questions. First, does varying the time interval between two consecutive irrigations modify acquisition, and second, how long can habituation established by repetitive caloric tests be retained?

1 Effect of the time interval between irrigations

This problem was investigated in the following manner. One ear of the cat was irrigated with water at 20°C once a day for ten consecutive days.

The results showed (Fig. 5) that the response decline of the eye velocity followed the same pattern as that obtained with repetitive irrigation at five minute intervals.

Commentary. This finding suggests strongly that the change in the vestibulo-ocular reflex arc produced by one irrigation was retained for at least 24 hours. The findings showed also that the cumulative effect of repetitive stimulation

in this case was as prominent as that in which the irrigations were applied at intervals of five minutes

The observations are of practical importance for studies of the cat's vestibular function. Response decline resulting from repetitive caloric stimulation might be attributed erroneously to some other variable. This problem may not exist when carrying out vestibular tests in man. Tello & Morales (1958, 1959) examined the vestibular function of patients with neuro-otological disturbances. Caloric tests with water at 44°, 30° and 20°C applied to both ears were given three times a day. These patients received at least 18 irrigations within 24 hours. As far as we can judge by their figures and discussion, no habituation occurred. These findings (Tello & Morales 1958, 1959) agree with the experience of Hood & Pfaltz (1954) and Cawthorne *et al.* (1956) in man. Hamersma (1957) and Lock & Haines (1946) on the other hand did report a response decline with repetitive caloric tests in man.

Because of the practical importance of nystagmography in the clinic, the question of whether habituation to caloric stimulation occurs in man should be further explored.

2 Persistence of habituation

This series included several animals which were habituated with one trial (water of 20°C) in one ear. After several days of rest, the same ear was tested again with water at 20°C.

The results revealed variability among animals which is illustrated in Fig. 6. Cat A 894 represents those cases, although rare, of recovery after two days of rest. Cat A 900 and A 897 belong to a group which retained habituation for two or more days. Most of these animals showed a progressive response decline. Within this group, however, a few exhibited recovery four or five days after the original stimulation. In all animals that showed partial or complete recovery, one or more additional trials served to re-establish habituation.

Further determinations on the persistence of response decline were made only occasionally because when testing for the presence of habituation, the tests themselves influence the results. In one animal, response decline was still found after three weeks of rest, with three months of rest, nystagmic responses were normal in another animal.

Commentary. Although these experiments did not study extensively the phenomenon of retention, nonetheless the results indicate that in the cat the response decline may persist for two or three weeks.

C Transfer of Habituation

Habituation to repetitive caloric stimulation and testing for transfer can be carried out under several conditions. For instance, the test for transfer of that habituation originally established by presenting hot water to one

The results demonstrated consistently that tests with equivalent stimuli in the left ear showed a marked response decline while nonequivalent stimuli applied to either exposed or nonexposed ears provoked normal responses. In several animals however both eye velocity and duration of nystagmus were somewhat diminished when tested with nonequivalent stimuli. For example Fig. 8 shows a case in which a trial with water at 20°C in the right ear produced a clear cut response decline. The transfer of habituation was evident when the left ear was tested with an equivalent stimulus but testing of the right and left ears with nonequivalent stimuli i.e. water at 48°C and water at 20°C respectively gave responses which were diminished relative to that shown at the top of the figure. A factor which may influence the comparison between acquisition of habituation and testing of transfer is that the strength of the stimulus with water at 48°C is less than that with water at 20°C.

In addition to this series three animals received at intervals of three or more days trials with water at 20°C in the right ear. Tests for retention of habituation were often given between trials. When extinction or pronounced response decline was obtained then the left ear was tested for transfer with water at 20°C. The results as shown in Fig. 9 demonstrated that no transfer occurred. Notice that the response of the left ear of Cat A 900 when tested with nonequivalent stimuli is diminished in the same way as that shown in Fig. 8.

Commentary These experiments established that transfer is demonstrated mainly when the nonexposed ear is irrigated with equivalent stimuli. The response decline of this ear indicates that the neural mechanism responsible for habituation of nystagmus is a central process. The evidence suggests strongly that neither adaptation nor fatigue of the vestibular receptors is the cause of acquisition of response decline. The fact that transfer of habituation is demonstrated in the ear non exposed when it is properly stimulated i.e. with an equivalent stimulus indicates that the neural mechanism responsible for the direction of the nystagmus is probably the locus of the phenomenon.

(c) Several animals were habituated with nonequivalent stimuli in two manners. One the right ear received one trial consisting of cold water irrigations alternating with hot water. The left ear was tested for transfer with both hot and cold water. Two the right and left ears were irrigated alternately with cold water. Testing for transfer was done with hot water in both ears.

It should be noticed that in both procedures the nystagmus during habituation was bidirectional and consequently each habituating stimulus had its equivalent counterpart in one of the stimuli presented during transfer tests.

The results with either procedures revealed complete transfer of habituation (Fig. 10).

Commentary In these cases transfer is due to the fact that nystagmus both toward the right and left was habituated. For instance in the case represented in Fig. 10 the right ear was irrigated with cold and hot water. Cold water produced nystagmus toward the left and according to the results of experi-

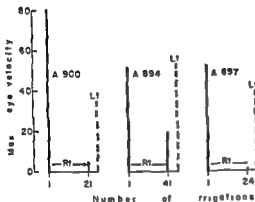


FIG 9

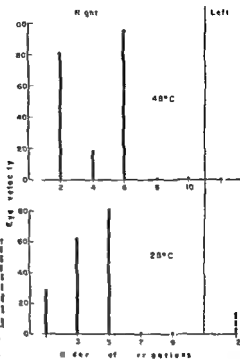


FIG 10

FIG 9 Transfer of habituation. These three cats received numerous irrigations as indicated in the figure with water at 20°C in the right ear resulting in nystagmus toward the left. Reversing the direction of nystagmus by irrigating the left ear with water at 20°C showed no transfer of habituation.

FIG 10 Transfer of habituation. Cat A 939. The right ear was irrigated with water at 28°C (lower part of the figure) alternating with water at 48°C (upper part). Extinction of responses was obtained to both cold and hot calorization. Irrigation of the left ear with either hot or cold water revealed transfer of habituation.

ment (b) it is expected that when testing the left ear with water at 48°C a response decline should be obtained. The same explanation holds for the response decline in the left ear when tested with cold water. The results obtained with the second procedure are similarly explained.

(d) It has been demonstrated that no transfer of habituation occurred to tests with nonequivalent stimuli when equivalent stimuli were presented as a trial alternately to the right and left ears. This observation was further investigated by applying the equivalent stimuli simultaneously. The animals received one trial with water at 48°C in the right ear and 28°C in the left. These simultaneous irrigations always produced horizontal nystagmus toward the right. After completion of the trial a Fitzcerald-Hallpike caloric test was conducted to find out whether transfer of habituation was present.

The results of testing showed (Fig. 11) normal responses when the nystagmus was provoked toward the left.

Commentary. The consequence of simultaneous irrigation of both ears

with equivalent stimuli are different from those produced by rotary stimulation even though in each case both ears are being stimulated at the same time. Although the pattern of habituation produced by both repetitive caloric and rotary tests are quite similar transfer as tested with nonequivalent stimuli does not occur under the former stimulating condition. This is due to differences in delivering the stimuli to the vestibular receptors. In the caloric test the stimuli are presented simultaneously in both ears only once in the course of each irrigation. With each rotation on the other hand the stimuli are presented twice i.e. one during acceleration and again with deceleration. This provokes nystagmus in one direction and then the other which is called per rotatory and post rotatory respectively. So during a repetitive rotary test in one direction both per rotatory and post rotatory nystagmus are habituated. Consequently response decline is expected when the direction of the rotary test is reversed.

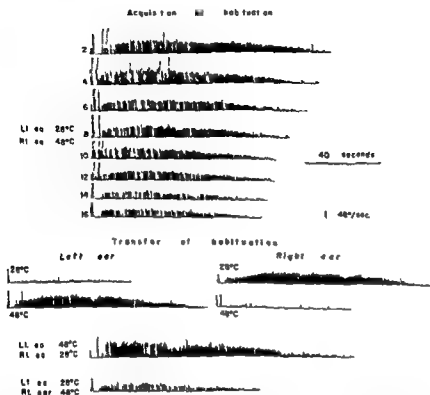
D. Final Remarks

It should be clearly understood that habituation of vestibular nystagmus is one behavioral event among several which tend to disappear with repetitive elicitations. This phenomenon which has been observed throughout the animal scale seems to represent a fundamental property of living matter. Halstead (1933), Humphrey (1933), Hilgard (1931), Thorpe (1930) and others pointed out that the characteristics of habituation are essentially similar to those of learning.

The vestibular reflexes playing an important role in the maintenance of body posture are constantly being stimulated under normal conditions without signs of habituation. It seems as if suppression of these reflexes occurs when the magnitude of the stimulus is above normal physiological values. Certainly this is the case with both rotary and caloric tests. A dramatic example of habituation to strong vestibular stimuli is shown by the performances of figure skaters, acrobats and flyers. McCabe (1960) observed that in figure skaters not only the vestibulo-ocular reflex arc is suppressed but also the postural reflexes.

Our experiments support the opinion (Fearing 1941; Hood & Balz 1954; Mowrer 1934) that neither fatigue nor adaptation of vestibular receptors cause the response decline. It is apparently due to a central process the nature and locus of which are not yet known. The cerebellum may participate in this process (Halstead 1933; Halstead, Yacorzanski & Fearing 1937) but perhaps the reticular formation and other structures also play a fundamental role. Further information of the neural process underlying habituation would well lead to a better understanding of higher functions of the central nervous system.

The experimental work on the response decline of nystagmus is based upon stimulation of the receptors located in the horizontal canals. The question remains whether other types of stimulation such as electrical



1 to 11 Transfer of habituation. Cat A 966. This animal received one trial consisting of 16 irrigations. The left and right ears were irrigated simultaneously, the former with water at 28°C, the latter with water at 48°C. The nystagmus was towards the right. Only records from even number irrigations in the total sequence of acquisition are included. Notice strong response decline to irrigation No. 16. The Fitzgerald Hallpike caloric test revealed habituation of nystagmus toward the right. This test also shows a characteristic directional preponderance toward the left. The last two records were obtained from simultaneous irrigations presented in the manner indicated ahead of the tracings. They show that the nystagmic responses toward the left are normal while those toward the right are diminished. The starting point of all tracings coincides with the completion of the caloricization.

may also produce habituation. Another important question, not yet explored, is whether stimulation of the other vestibular receptors will produce habituation. There is no compelling reason to expect that this would not occur. Unfortunately, little is known about habituation of vestibular reflexes other than nystagmus. Apropos of this point, we never observed spontaneous nystagmus and/or disturbances of equilibrium after the nystagmic response was extinguished.

A thorough investigation of the conditions under which habituation of vestibular reflexes take place in man is important for practical reasons. First, the results would be of considerable significance for interpreting repetitive rotary or caloric tests as used in clinics (Hood & Pfaltz, 1954). Second, habituation of vestibular reflexes may be a useful tool for improving

the performance of special tasks requiring compensation for extreme movements. Third, compensation for vestibular disorders may well be accelerated by a judicious use of the response decline induced by repetitive vestibular stimulation.

ZUSAMMENFASSUNG

Der Vorgang des Gewöhnens der Katze an sich wiederholende kalorische Reizungen wurde untersucht. Die Untersuchung umfasste Erwerbung Beibehalten und Übertragung der abnehmenden Empfindlichkeit in nystagmischen Reaktionen. Kalorisches Gewöhnen wurde durch wiederholte Kalorisation des rechten und/oder linken Ohres mit kaltem und/oder heissem Wasser hervorgerufen. Die Empfindlichkeit nahm während der einleitenden Spülungen schnell und später langsam ab. Der Umfang der Empfindlichkeitsabnahme war in den verschiedenen Versuchstieren verschieden und reichte von einer gemässigten Abnahme bis zu einer völligen Auslöschung jeglicher Empfindlichkeit.

Gewöhnung kann in der Katze nach drei Wochen Ruhe beobachtet werden. In einigen Tieren aber stellte sich normale kalorische Empfindlichkeit schon nach wenigen Tagen wieder her.

Übertragung des Gewöhnens findet sich, nur wenn der Nystagmus in derselben Richtung hervorgerufen wird in welcher habituiert wurde.

Die Ergebnisse der Untersuchung zeigen, dass der neurale Mechanismus welcher der Gewöhnung des vestibulären Reflexes zugrunde liegt, ein zentraler Vorgang ist. Der Angriffspunkt dieses Mechanismus bleibt unbekannt. Die praktischen und theoretischen Folgerungen dieses Vorganges werden kurz besprochen.

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STUDIES ON INNER EAR FUNCTION AND CRANIAL NERVES IN DIABETICS

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Results of an oto-neurological study comprising 69 diabetics are reported. An inner ear lesion was found in 28 cases characterized as a rule by progressing symmetric hearing loss of the perceptive type. A few patients reported acute onset of hearing loss accompanied by tinnitus and dizziness. In cases where recruitment tests could be carried out the disease was found to be located in the cochlea. Vestibular function tests did not reveal involvement of the vestibular part of the labyrinth. One of the patients had peripheral facial palsy. A correlation was found between the inner ear lesion and the late diabetic complications as indicated by the degree of retinopathy. Hearing loss was commoner in elderly than in younger diabetics. The pathogenesis is assumed to be angiopathy affecting the blood vessels of the labyrinth or acoustic nerve similar to that which may be found in other late complications of diabetes.

The discussion on the relationship between diabetes mellitus and diseases of the acoustic and vestibular organ has been going on for more than 100 years. Jordan in 1857 was the first to report a case of incipient diabetic coma in a 41 year old man exhibiting impairment of vision, hearing, smell and taste. Griesinger (1859) and Kulz (1874) each reported three cases of labyrinthine disorders in diabetic subjects. The chief sign was impairment of hearing but a few also suffered from tinnitus and vertigo. Foynble in 1860 published the autopsy findings in a 29 year old diabetic who had died of necrotizing mastoiditis complicated by perisinuous abscess but he did not relate these findings to the diabetes. Ravnaud (1881) set up the concept *Otitis diabetique* characterized by a greatly haemorrhagic discharge and a fulminant course describing this syndrome in a patient with severe diabetes. The post mortem including gross inspection of the temporal bones had been carried out by Ladreit de Lacharrière. A similar syndrome was described in Germany by Wolff (1887) who called it *otitis media necroticans sine diabetica*.

Interest soon centred on the inflammatory diseases of the external and middle ear probably because these diseases spelt immediate danger to life and a vast literature soon accumulated. Meanwhile interest in the inner ear affection dwindled. In Senator's (1876) opinion impairment of hearing was a rare associated phenomenon whereas tinnitus was commoner.

Gruber (1888) in his textbook stated that diabetes might be accompanied by morbid changes of the inner ear characterized by impairment of hearing and tinnitus, whereas Steinbrugge (1901) in Orth's textbook makes no mention of the inner ear affection. Fulaenstein (1899) did not accept the presence of any inner ear disorder in diabetics and did not feel that even inflammatory aural diseases ever ran a course characteristic of diabetes mellitus.

In 1899 Kulz published his comprehensive study of 692 diabetics. Fifty-nine (8.5%) had aural symptoms. The only demonstrable cause of the hearing loss was the diabetes in 22 patients after patients with complicating diseases such as syphilis, pulmonary tuberculosis, etc. had been excluded. He had demanded normal otoscopic findings and an onset of hearing loss after diabetes mellitus had been diagnosed.

In his large monograph on diabetes von Noorden (1912) concluded that in diabetics pure hearing loss without other abnormal findings was commoner than otitis media.

The first systematic investigation on inner ear function in patients with diabetes mellitus was performed by Edgar (1915). He tested the hearing of 52 patients for whispered and spoken voice, did tuning fork tests including Rinne's, Weber's and Schwabach's, and a few patients were tested by Bezold's continuous series of tuning forks. The upper tone limit was investigated by a high frequency tuning fork and Galton's pipe. Vestibular function was tested by rotation tests, caloric and galvanic stimulation experiments being carried out in only one case. In 27 out of the 52 patients Edgar found bilateral hearing impairment of the perceptive type, mainly for high tones. In a few cases he found disturbances of balance, but only one patient had nystagmus of a mild degree. Three showed a tendency to fall in the Romberg test. Moreover, the hearing impairment proved dependent upon the severity and duration of the diabetes, and it had usually developed slowly without the patient noticing. In a number of those patients who also had fairly severe arteriosclerosis, annoying tinnitus was often present.

For many years Edgar's study remained the only major systematic investigation into the subject, and Federer's (1926) contribution on the diseases of the inner ear in diabetes in Denker & Kahler's *Handbuch der Hals-, Nasen- und Ohrenheilkunde* was mainly based on Edgar's studies. He stated that diabetic inner ear disease was of insidious onset, of an extremely chronic course, and generally bilateral. However, unilateral labyrinthine damage has been reported. Hegener (1908) for instance described the case of a diabetic who developed attacks of vertigo, tinnitus, and hearing impairment of the perceptive type in the left ear. This subsided, leaving mild hearing loss of high tones.

Isolated affection of the vestibular apparatus has been described by Tang (1917). In a male diabetic, aged 61, who was complaining of vertigo when changing the position of his head, he found hypersensitivity of the left vestibular apparatus upon caloric test by the method of Rutlin. In this case, the symptoms disappeared after two weeks' antidiabetic therapy.

In *Handbuch der Neurologie des Ohres* Benesi & Sommer (1929) also refer mainly to Edgar's results but add five cases of their own of labyrinthine lesions in diabetics. In their cases too the main symptom was high tone hearing loss of the perceptive type. Moreover three of the patients complained of tinnitus. Two were suffering from gyratory vertigo. In one of them vestibular function studies showed normal reactions to rotation but bilateral weakening of the caloric reaction. This patient also had left sided ataxia and adiadochokinesia.

After the advent of insulin in the treatment of diabetes in the nineteen twenties the outlook in this disease was radically changed for the better and many years could be added to the patients' lives. This improvement in longevity was further accelerated after the introduction of sulphonamides and antibiotics in the treatment of the previously dreaded complications to infections in diabetes. Thus while previously interest was focussed chiefly on the acute and often tragic terminal complications of the disease more attention has been paid in the past few decades to diabetic late complications which of course have been observed with an ever increasing frequency.

Owing to the insidious and chronic course of diabetic inner ear affection one would expect an increasing incidence of this complication. The first otologists displaying an interest in this aspect were Jannulis & Deljaninis (1936) who studied 74 diabetics finding 43 (58%) to have acoustic lesions due exclusively to the diabetes in 29 (39%). Their hearing was not tested by audiometry but with whispered voice spoken voice and the Weber, Rinne and Schwabach tests. Hearing loss was most marked for high tones and of the perception types. In some of the patients it had come on rather suddenly but in most of them the onset had extended over several years. No relationship was found between the duration and severity of the diabetes on the one hand and the hearing impairment on the other. There were mild cases with severe hearing loss and severe cases with only slight hearing loss. Some of the patients were suffering from tinnitus and others from vertigo. Rotatory and caloric function tests however did not reveal any definite damage to the vestibular organ.

In Joslin's book *The Treatment of Diabetes Mellitus* Root (1946) states that Meniere-like symptoms are a common and annoying complication especially in middle aged and elderly diabetics. He mentions two examples. A 44 year old woman with a 20 year history of diabetes suddenly got aural pain, vertigo and unilateral deafness. Another patient a 47 year old woman with a history of diabetes for eight years developed a genuine Meniere syndrome successfully treated by operation. In the more recent editions of Joslin's book inner ear affection is not mentioned. In our opinion Root's first example cannot be interpreted as Meniere's disease since in this disease total and irreversible deafness does not usually occur after the first attack.

The first audiometric measurements of the hearing were done in the fifties. (Jannissica (1950) examined 81 diabetics ranging in age from 29 to 75 years. In 13 he found normal hearing while 37 had hearing loss of the perceptive

type nine of the conduction type and two hearing loss which could be put down to acoustic trauma. In the few cases in which the hearing loss was not bilateral and symmetric Fowler's test revealed recruitment indicating a cochlear location of the inner ear affection. A correlation was found between the degree of hearing loss on the one hand and the duration and severity of the diabetes on the other. Vigi also in 1950 examined 150 diabetics divided into three groups viz. patients under 30 years of age (11) patients between 30 and 50 (30) and patients over 50 years of age (109). All the patients of the first group had normal hearing. In group 2 mild hearing impairment was found in 20% and there was no case of severe hearing loss. Within group 3 19% had normal hearing while 39% had moderate and 43% severe hearing loss. The audiometric curves were typical of perceptive hearing loss of the senile type. Among 100 diabetics Marullo (1950) found 47 to have perceptive deafness of the senile type. The degree of hearing loss proved to be correlated to blood pressure but not to the severity of diabetes in terms of the hyperglycaemia.

Borsuk (1950) found among 108 patients 35 with abnormal audiograms characterized by hearing loss of high frequencies. There was a correlation between the degree of hearing impairment and the duration and severity of the diabetes. Ancona in 1956 examined 27 patients aged 16 to 33 years with a history of diabetes since childhood. Thirty five per cent showed bilateral symmetrical hearing loss of high tones of the perceptive type. This worker found no relationship to the severity and duration of diabetes. He mentions that the recruitment phenomenon was not demonstrable but does not mention his technique although all the measured hearing losses were symmetrical.

In Schroder's (1954) opinion there is no reason to assume that diabetes attacks the acoustic nerve as he found no characteristic hearing impairment among 217 patients from the Berlin diabetes clinic compared with a normal series of 300 patients. However only 10% of these patients were examined by audiometry. Kindler (1955) also does not believe there is a special diabetic inner ear affection. He refers to Schroder's investigations stating that he was unable to find a single case of hearing impairment among diabetics unless it was explicable by the common diabetic complication of premature arteriosclerosis. In order to study this question in more detail Profazio & Baravelli (1959) examined 40 diabetics who required insulin comparing the audiometric curves with Johansen's (1943) normal series in order to exclude hearing loss of the senile type. Hearing loss was not found in patients under 44 years of age. Hearing loss apart from presbycusis was present in 26% of the patients aged 51-60 and in 37% of the patients aged 61-70 years.

Only a few recent studies have been concerned with the vestibular apparatus. Cammiser (1960) found normal vestibular reactions in 48 out of 71 patients who had the Veit's caloric test. Twenty two had bilateral hyporeflexibility, nine bilateral hyperreflexibility and 17 asymmetric reactions. There was no relation to the severity of the diabetes. Cozzazzi (1950) studied

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putting forward the concept *neuritis degenerativa cochlearis atrophicans diabetica* for which however there are no pathognomonic symptoms. Hegener (1908) is the only author who has suggested that the not uncommon episodes of sudden hearing impairment, tinnitus and vertigo in diabetics might be explicable by minor labyrinthine haemorrhages.

From the above review of the literature it will be seen that according to most workers diabetics may have an inner ear affection characterized by slowly usually bilaterally and symmetrically progressive hearing loss which is difficult to distinguish audiometrically from senile or arteriosclerotic hearing impairment found in normal subjects. Comparing the audiometric findings in diabetics and a normal series one author found a significant difference in the form of a more severe hearing loss among diabetics. In a few cases of asymmetric hearing loss the lesion has been located to the cochlea by the Fowler recruitment test. In addition to the chronic progressive and bilateral hearing loss a few cases of acute usually unilateral inner ear affections have been described with predominance of the cochlear component (hearing impairment and tinnitus) or of the vestibular component (vertigo). Some authors have related these attacks to Ménière's disease and one interpreted them as being possibly due to labyrinthine haemorrhages. These episodes are characterized by being transitory but possibly leaving sequelae in the form of unilateral hearing impairment and/or reduced vestibular irritability. Studies of vestibular function have not revealed any characteristics, some workers having found normal reactions and others a large number of abnormal reactions upon caloric stimulation.

Pathological studies are so few and incomplete that they do not allow any deductions.

At the Otolological Department of the University Hospital, Copenhagen the symptoms and pathogenesis of diabetic inner ear affection have been submitted to further clinical and pathological investigation. Below the results of the clinical studies will be reported.

MATERIAL

The material comprises 69 patients. Twenty three were referred to us from the Steno Memorial Hospital and 26 from the Ante natal Clinic of our hospital. As there is a female preponderance nor is the material representative of the age distribution of diabetics as we have deliberately examined as many young patients as possible in order to avoid the interference of physiological changes of aging.

A careful history was taken regarding the duration and severity of the diabetes as well as the occurrence of diseases of the ear, nose and throat. As far as the diabetes was concerned we recorded the following: age, duration of diabetes and its treatment as well as difficulties in controlling the diabetes as indicated by the number of episodes involving coma and insulin reactions. The patients were asked whether they had noticed fluctuations in

vision and hearing parallel to fluctuations in the blood sugar values and whether they had suffered from hearing impairment tinnitus or vertigo. Moreover they were questioned regarding previous otitis acute or chronic acoustic traumas or other incidents which may involve hearing loss.

The case records were searched for data regarding late complications of diabetes. All the patients had ophthalmoscopy and the retinal findings were classified as follows:

No retinopathy	0
Microaneurysms	+
Haemorrhages with or without exudation	++
Proliferative changes	+++

The nephropathy was assessed on the basis of the proteinuria and serum creatinine values. No graduation was attempted however neither of the nephropathy neuropathy nor of the angiopathy found in a few cases in the blood vessels of the lower limbs. Determination of the systolic and diastolic blood pressure was done in all cases but one.

In the Ear Clinic the patients had in addition to an ordinary objective examination of the ears nose and throat also an oto-neurological study as complete as possible. Their hearing was determined by audiometry including air and bone conduction in all cases. A few had a test for recruitment according to the Fowler method or by means of acoustic impedance as described by Metz (1952). Vestibular function was assessed by investigating the patients for spontaneous nystagmus and postural nystagmus as well as by a differential caloric test by the Hallpike method. Cerebellar function was studied by making the patients walk with closed eyes by Barany's pointing test and by a study for adiadochokinesia and dysmetria. The motor function of the 3rd 4th 6th 7th 10th 11th and 12th cranial nerves was studied by ordinary inspection of the patient and laryngoscopy. Trigeminal sensitivity was studied for differences in the sense of pain and touch on the left and right including corneal sensitivity assessed quantitatively by the Boberg Ans (1952) apparatus. Moreover the sense of taste and smell was investigated qualitatively as well as quantitatively. The results of these last mentioned studies will be published in a subsequent paper.

RESULTS

Table 1 gives the results of the audiometric measurements related to the patients diabetes and possible late complications. The patients are listed by age. It will be seen that hearing loss was found in 79 cases. In order to gain an impression of the number of cases in which the hearing loss could be ascribed with fair certainty to the diabetes cases in which the hearing loss was near physical normal were ruled out. This was done by comparing in each individual case the hearing losses found in our patients with the hearing loss found by Jørgensen (1943) in the corresponding age group of his normal

TABLE 1. *Hearing loss related to other diabetic manifestations in 69 patients*

Case no	Sex	Age of patient	Duration of diabetes	Duration of insulin treatment	Diabetes easy to control	Diabetes difficult to control	Retinopathy	Nephropathy	Neuropathy	Vascular manifestations in legs	Blood pressure	Hearing loss
1	F	16	7	7	+		-	-	+	-	120/75	+
2	F	16	■	5	+		■	-	-	-	125/85	(+)
3	F	17	12	12		+	■	-	-	-	110/80	-
4	M	17	12	12		+	■	-	-	-	120/80	-
5	F	17	■	9		+	■	-	-	-	125/75	-
■	F	19	9	9	-		0	+	-	-	110/75	-
7	F	19	11	11	+		+	-	-	-	140/110	-
8	F	20	■	6	+		+	+	-	-	125/90	-
9	F	20	14	14	+		0	-	+	-	110/75	-
10	F	21	4	4	+		0	-	-	-	115/80	+
11	F	■	15	15		+	+	-	-	-	130/80	-
12	F	22	16	16	+		+	+	-	-	135/100	+
13	F	23	15	15	+		+	-	-	-	120/90	-
14	F	23	16	16	+		+	+	-	-	125/80	+
15	F	23	8	8		+	0	-	+	-	115/80	-
16	F	23	17	17	+		+	+	-	-	120/80	-
17	F	24	4	4	+		(+)	+	-	-	125/90	+
18	F	25	13	13	-		+	+	-	-	120/70	-
19	M	25	9	9	+		+	-	-	-	100/60	(+)
20	F	26	11	11	-		+	-	-	-	130/80	-
21	F	26	15	15		+	+	-	-	-	140/80	+
22	F	27	1	1	+		0	-	-	-	110/65	-
23	F	27	14	14	+		■	-	-	-	115/80	-
24	F	27	3	3	+		0	-	-	-	110/60	-
25	F	27	5	5			0	-	-	-	140/70	(+)
26	F	28	9	9	+		0	-	-	-	130/70	-
27	F	28	26	26		+	0	-	-	-	120/80	(+)
28	F	28	4	4	+		■	-	-	-	125/85	-
29	F	28	14	14		+	0	-	-	-	110/65	(+)
30	F	28	13	13	-		+	+	+	-	120/90	-
31	F	30	15	15			0	-	-	-	160/100	+
32	F	30	22	22	-		-	+	-	-	135/90	-
33	F	31	15	15		+	+	+	-	-	160/100	+
34	F	31	3	3			0	-	-	-	120/80	-
35	M	31	19	19		+	+	+	+	-	115/75	(+)
36	M	31	27	27			-	-	-	-	125/75	+
37	F	31	18	18	+			+	+	-	115/80	-
38	M	31	32	32		+	+	+	-	-	115/80	+
39	F	34	1	1	+		0	-	-	-	110/65	-
40	F	36	18	18		+	0	-	-	-	120/80	-
41	M	36	21	21	+		+	+	-	-	120/80	+
42	F	36	30	30			+	-	-	-	115/80	+
43	F	39	■	■	+		0	-	-	-	120/80	-

vision and hearing parallel to fluctuations in the blood sugar values and whether they had suffered from hearing impairment tinnitus or vertigo. Moreover they were questioned regarding previous otitis acute or chronic acoustic trauma or other incidents which may involve hearing loss.

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TABLE 2 *Hearing loss in decibels in 28 diabetic patients*

Case no Age of patient	125 r l	250 r l	500 r l	1000 r l	2000 r l	4000 r l	8000 r l
1 16	20 30	10 25	10 20	5 20	5 10	0 5	10 25
10 21	5 5	0 0	0 0	0 0	0 0	0 0	20 25
12 22	0 0	0 0	0 10	0 20	10 20	20 20	30 20
14 24	0 0	0 0	0 0	0 0	5 0	5 10	20 30
17 24	0 10	0 10	0 5	0 5	0 10	5 10	5 50
21 26	25 20	20 20	20 20	0 20	0 20	10 20	20 40
31 30	20 40	20 30	20 20	20 20	0 20	10 20	20 40
33 31	20 25	20 25	20 20	40 20	>100 20	>100 15	60 40
36 31	20 10	10 10	10 10	0 0	20 20	30 30	30 40
38 34	20 0	20 0	40 0	40 20	40 20	25 10	0 0
40 36	20 20	20 0	10 10	20 0	20 10	10 0	20 20
41 36	60 40	80 40	80 60	80 80	60 80	60 60	80 80
42 36	10 0	10 0	10 0	10 0	5 0	5 0	40 0
44 39	0 0	10 10	0 10	10 10	20 20	25 20	20 20
49 45	20 20	20 20	20 20	10 20	20 30	20 25	20 35
50 47	20 10	20 10	10 10	20 10	20 10	60 10	80 60
51 48	20 40	20 20	30 40	30 30	30 45	45 45	60 50
52 48	20 10	20 5	5 10	5 5	20 30	40 30	40 40
56 50	40 20	30 20	20 0	20 0	20 30	20 30	30 60
57 53	40 20	40 20	45 40	45 40	40 50	50 40	70 60
59 59	10 20	10 20	10 25	20 20	10 30	60 50	80 >100
61 59	20 30	20 20	10 20	0 0	20 20	20 20	20 40
62 59	20 20	20 10	20 20	20 20	10 40	20 30	30 20
63 60	20 40	20 40	30 40	20 30	10 40	40 40	60 60
65 64	20 20	40 20	40 50	20 30	30 40	100 100	90 100
66 68	40 40	30 20	30 20	30 20	40 40	60 60	80 >80
67 70	40 60	40 60	40 60	40 30	25 25	30 20	70 60
69 73	60 20	60 40	60 40	60 40	60 60	80 60	90 80

affect not only the high frequencies but several patients may show marked hearing loss of the deep tones as well. There is no distinct correlation between the duration of diabetes and the impairment of hearing while as shown in Table 3 hearing loss is more often encountered in elderly than in younger patients.

Table 4 shows the correlation between the severity of retinopathy and the hearing impairment hearing loss being twice as common in patients with severe proliferative retinopathy as in patients without retinopathy as in patients without retinopathy. In patients under 40 years of age there is an unmistakable correlation between nephropathy and hearing loss seven out of 14 patients with nephropathy having hearing loss which was present in only six out of 30 without nephropathy. This correlation was not found in patients over 40 years of age. In the present series no correlation between hearing impairment and diabetic neuropathy was demonstrable. Also, there

TABLE 3 *Hearing loss in the different age groups*

Age group	Total	With hearing loss
0-20	7	1 (14%)
21-30	23	5 (22%)
30-40	11	8 (58%)
40-50	9	1 (11%)
50-60	9	5 (55%)
Over 60	7	5 (71%)
	69	28

TABLE 4 *Hearing loss in relation to retinopathy*

Retinopathy	No. of cases	Cases with hearing loss
0	31	9 (29%)
+	11	5 (45%)
++	18	9 (50%)
+++	9	5 (55%)
	69	28

was no correlation between hearing impairment and blood pressure values. In the great majority of cases the hearing loss had developed slowly without the patients noticing but in the three cases with the most severe hearing loss (Cases 33, 41, and 69) the hearing loss had come on suddenly and had been accompanied by transitory tinnitus. One of these patients was also suffering from attacks of vertigo. These three patients had had their diabetes for 15, 27, and 13 years respectively, and all had severe proliferative retinopathy. Cases 33 and 41 also had retinal haemorrhages. Their histories have been published previously (Jørgensen 1960). Case 69 was suffering from severe diabetic angiopathy affecting the lower limbs, and for this reason the right leg had been amputated 4 years previously. Recruitment tests were carried out in Cases 41 and 69. On the basis of the findings, the hearing loss was traced to the cochlea in both. Symptoms in the form of tinnitus were present in only six cases, viz. Cases 44, 57, and 67 in addition to the three mentioned above. All the patients having tinnitus were of the group with hearing impairment. Two had arterial hypertension.

Diabetics often complain of fluctuations in their visual acuity parallel to fluctuations in the blood sugar values, presumably due to alterations in the osmotic tension in the eye. This applied to 23 out of 69 patients. As far as hearing was concerned, this was found in only one patient (Case 43) who complained of impaired vision and hearing on the left during periods of hypoglycaemia.

TABLE 5 Findings in 9 patients complaining of vertigo

Case no	Age of patient	Duration of diabetes	Arterial hypertension	Hearing loss	Spontaneous or positional nystagmus	Abnormal caloric reactions
33	31	15	+	+	0	0
40	36	18	0	+	0	0
44	39	30	0	+	0	0
55	59	8	+	0	+	+
61	59	3	+	+	0	0
64	60	6	0	0	+	0
66	68	5	0	+	0	0
67	70	5	0	+	0	0
69	3	13	+	+	0	0

Nine patients (Case 33 40 44 55 61 64 66 67 and 69) complained of dizziness. Seven belonged to the group having hearing impairment and four of them were suffering from hypertension. While in the three patients who were under 40 years of age the dizziness occurred after a long history of diabetes, there was no correlation between the vertigo and the duration of diabetes in the six patients over 40 years of age. There was also no correlation between vertigo and the severity of retinopathy. Vestibular function tests performed in all 69 cases showed abnormal reactions in only two. Case 55 had spontaneous nystagmus to the right and the differential caloric test revealed a preponderance to the right. This patient's hearing was normal and the blood pressure 190/110, so the dizziness was assumed to be of central origin. Case 64 had severe postural nystagmus to the left when lying on her right side. No abnormality was found in the differential caloric test. This patient was obese and suffering from mild diabetes which could be controlled by diet alone. Her hearing and blood pressure were normal. In this case too the dizziness was assumed to be of central origin. Table 5 gives the age, duration of diabetes, blood pressure and objective findings in the acoustic vestibular apparatus in the nine patients who complained of dizziness.

All tests of cerebellar function gave normal results.

Investigation of the cranial nerves showed normal trigeminal and corneal sensitivity in all cases. The function of the motor nerves proved to be normal in all cases but one. Case 42 who had had left sided peripheral facial palsy for 4 years. This patient was 36 years of age and had been suffering for the past 30 years from diabetes which had always been difficult to control. However, she did not have late complications apart from mild retinopathy and a slightly impaired hearing.

COMMENTS

In a series of diabetics we found in accordance with previous authors cases of hearing impairment of the perceptive type correlated to the severity

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KNOCHENLEITUNGS-SPRACHAUDIOMETRIE ALS ZUSÄTZLICHE MESSMETHODE VOR HÖRVERBESSERNDEN OPERATIONEN¹

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Eine Zusammenfassung befindet sich am Ende dieses Aufsatzes

Bei allen Prüfungen mit Eintonen, also mit Stimmgabeln und Audiometern, kann es vorkommen, dass Schwerhörige zwischen echtem Hören und nur taktil empfundenen Schwingungen nicht unterscheiden können. Dieses sogenannte „Hörfühlen“ im unteren Frequenzbereich kann zu Auswertungsfehlern im Audiogramm führen und hat u. U. eine falsche Indikationsstellung zur Operation zur Folge.

Auf S. 18 des „Leitfadens der praktischen Audiometrie“ bringt Langenbeck eine Kurve über das Schallfühlen Totaltauber in Luftleitung (1). Zeichnen wir in diese Abbildung noch die Knochenleitung doppelseitig Tauber ein, wie sie auch Oeken vergleichend auf dem Mastoid (2) und an der Fingerbeere (3) gemessen hat, so erhalten wir Abb. 1.

Wenn man weiter bedenkt, dass das Knochenleitungstelefon nur selten durch ein Stirnband mit einem genau definierten Auflagedruck (250 g/cm²) auf das Mastoid gedrückt wird, sondern bei der Routinemessung vom Probanden selbst (mit den sehr viel empfindlicheren Fingern) gehalten wird, so ist die Möglichkeit des Hörfühlens sehr gross. Zwar weist die Haut der Fingerbeere pro cm² wesentlich mehr Tastkörperchen auf als die des Mastoids, und es liegt nahe, auf Grund der beiden Zahlen auf die Empfindlichkeit zu schliessen. Nach Hensel haben aber die Haarrezeptoren in der Haut hinter dem Ohr eine völlig andere mechanische Empfindlichkeit als die Rezeptoren der Haut an der unbehaarten Fingerbeere. Im Vergleich zwischen beiden ist nur durch die Bestimmung der Vibrationsschwellen möglich. Oeken führte sie durch und fand bei Volltauben eine um 10–15 db bessere Fühlkurve am Zeigefinger als auf dem Mastoid. Die Prüfung der Knochenleitung kann bis 1000 Hz also gelegentlich nur Ausdruck einer Fühlschwelle der Tastkörperchen der Haut sein.

Im Hinblick auf hörverbessernde Operationen an hochgradig Schwerhörigen mit einer kombinierten Hörstörung ist diese Tatsache von grosser Bedeutung. Gerade bei diesem Personenkreis und insbesondere bei blinden Schwerhörigen sehen wir oft ein sehr ausgeprägtes Tastgefühl mit dement-

¹ Herrn Prof. Dr. med. B. Langenbeck in dankbarer Verehrung zum 63. Geburtstag gewidmet

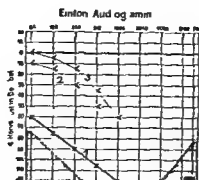


Abb 1 Föhlkurven bei Totaltauben in Luftleitung (1) und in Knochenleitung (Mastoid (2) Fingerbeere (3))

sprechend guter Knochenleitungsschwelle. Bekanntlich fällt der Rinne'sche Versuch mit tiefrequenten Stimmgabeln ($C_{64} H_7 - c^2 = 512 \text{ Hz}$) in derartigen Fällen oft negativ aus. Darum ist diese Prüfung auch nur mit der Stimmgabel $c^3 - 1024 \text{ Hz}$ wertvoll, da deren Stielton praktisch nicht gefühlt sondern tatsächlich nur gehört wird.

Bei der audiometrischen Prüfung mit Eintonen gibt der Proband Hören aber ebenso auch Horfühlen durch die Aussage Ja oder Nein an. Eine objektive Differenzierung zwischen echtem Hören und Horfühlen ist im unteren Frequenzbereich bis 1000 Hz nicht immer möglich. Sprachaudiometrisch lässt sich der Funktionszustand des Innenohres recht gut bestimmen. International ist es üblich einmal den Hörverlust für Sprache in Dezibel mit Zahlen oder Spondeln zum anderen den Diskriminationsverlust in Prozent mit Einsilbern zu messen. Von allen Autoren wurde Sprachaudiometrisch bisher aber ausschliesslich in Luftleitung gemessen. Zwar wiesen wir nach (Sprachaudiometrie Thieme Verlag 1957 und / Laryng Rhinol 39 (1960) 110 ff) dass bei Prüfung mit dem Zahlen- und Wertertest signifikante Kurvenbilder entstehen, die einmal für eine Schalleitung zum anderen für eine Innenohrstorung sprechen. So sehen wir auch bei der Durchsicht aller Sprachaudiogramme der bislang in unserer Klinik gefestigten Otosklerosekranken, dass bei überschüssender Knochenleitung im Eintonaudiogramm sprachaudiometrisch immer ein 100%iges Wortverständnis erreicht wurde.

Nachdem wir seit 7 Jahren Frommelfellplastiken und in den letzten Jahren zunehmend Stapesmobilisationen und Interpositionen durchführen, werden in grösserem Masse auch Schalleitungsschwerverhörige mit geringerer oder grosserer Innenohrkomponente operiert. Die diagnostische Klärung dieser vorgeschrittenen Fälle bereitet aber oft Schwierigkeiten und eine Voraussage über die postoperative Hörerwartung ist schwer zu stellen. Die uns in diesem Zusammenhang vor allem interessierenden Grenzfälle, also die hochgradig kombinierten Schwerhörigen, gehen mit den bisher üblichen Messmethoden uncharakteristische Kurvenbilder, so dass gelegentlich eine fehlerhafte Indikation für eine hörverbessernde Operation gestellt werden kann.

Sprachaudiogramm

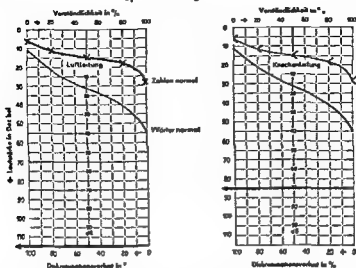
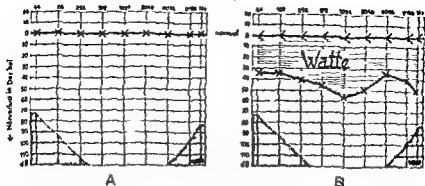


Abb. II Sprachaudiogramm eines Normalhorenden mit Zahlen geprüft. Links in Luftleitung (maximale Leistung 115 db) rechts in Knochenleitung (maximale Leistung 80 db). Um eine übersichtlichere Darstellung zu erreichen wurden die Wortkurven (PB) nicht abgebildet.

Bei Messungen mit der Zollner Thullen'schen Hörsonde stellten wir fest, dass die Probanden gelegentlich schon die taktile Berührung der Sonde im Mittelohr als Hörensation angaben und wir unrichtige Ergebnisse erhielten. Versuchsweise prüften wir daher mit der Sonde ausser mit Lintönen auch sprachaudiometrisch mit Zahlen. Bei fehlerfreiem Nachsprechen waren wir sicher, genaue Messpunkte erhalten zu haben. Lundborg führte in letzter Zeit klinische Messungen mit der Schallsonde durch, die von Rosler auch technisch weiter untersucht wurden. Die Konstanz der Messergebnisse wurde dabei durch Verwendung dickerer und gerader Sonden verbessert. Jedoch hielt auch diese Konstruktion Schwierigkeiten in Bezug auf den guten Einblick durch den Gehörgang und die Frequenzkurve ist auch noch nicht wünschenswert ausgeglichen. Es läuft deshalb in unserem Akustischen Labor eine Forschungsarbeit, bei der die Herren Dr. Michler und Dr. Lau die Brauchbarkeit in obengenanntem Sinne verbessern und ein physikalisch exakte Eichung und fortlaufende Kontrolle derselben sicherstellen wollen. Eine Schallsonde mit linearem Frequenzgang wurde auch eine einwandfreie Sprachübertragung ermöglichen, was mit den bisherigen Schallsonden nur sehr mangelhaft durchgeführt werden konnte. Zu unserem Erstaunen stellten wir nach orientierenden sprachaudiometrischen Messungen mit dem üblichen Knochenleitungstelefon auf dem Mastoid bei Otosklerosekranken fest, dass nicht nur einfache Zahlen sondern auch die schwierigen Einsilber des Freiburger Worttestes fehlerfrei gehört und nachgesprochen wurden. Im Rahmen einer Promotionsarbeit untersuchten wir mit Herrn cand. med. h. F. Leicht

¹ Sprachaudiometrie über Knochenleitung und Schallsonde zur exakteren Bestimmung der Innenohrfunktion. Dissertation Freiburg i. Br. 1940.

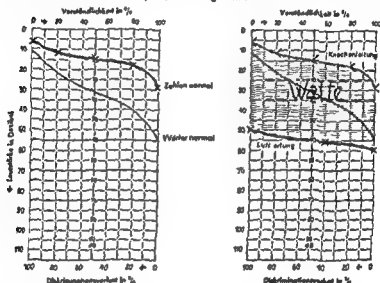
Einton Audiogramm



A

B

Sprach Audiogramm



C

D

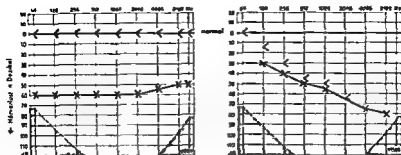
Von 3a-d B P 23 J Taubheit re nach Mittelohrfraktur mit Vestibularis und Acusticusausfall. Das normale Hörvermögen links wurde hier abgebildet: a) normales Eintonaudiogramm des linken Ohres b) experimentell erzeugte Schallleitungsschwerhörigkeit Hörverlust im Eintonaudiogramm 40-50 dB Knochenleitung normal c) normales Sprachaudiogramm in Luftleitung (nur Zahlen geprüft) d) das Sprachaudiogramm zeigt in Knochenleitung normale Werte und in Luftleitung einen Hörverlust von 10 dB. Beachte den korrespondierenden Hörverlust für Eintöne und Sprache bei Messung in Luft- und Knochenleitung in c und d.

eine größere Anzahl normal- und schwerhöriger Probanden vergleichend in Luft- und Knochenleitung sowohl mit Eintönen als auch mit Sprache. Nachträglich fanden wir in der Literatur, dass Goetzing & Proud 1955 und später Tato & Alfaro über ähnliche Untersuchungen berichtet haben.

Apparatur

In der camera silens (Storpegel unter 30 Phon bzw. 30 dB) prüften wir mit dem Atlas Sprachaudiometer I M48 in Luftleitung mit dem Becker Doppel-

Enton Aud ogramm



Sprach Aud ogramm

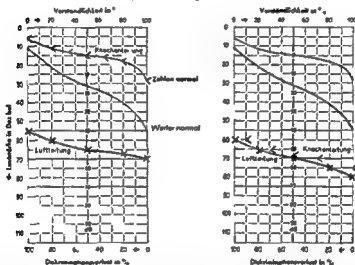
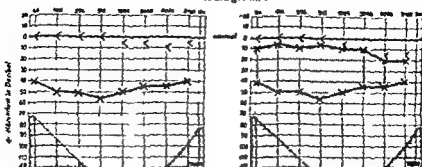


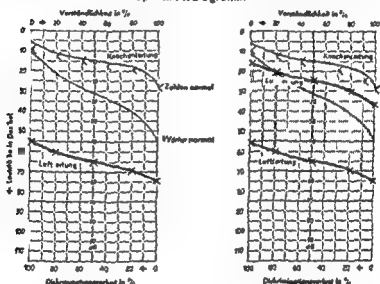
Abb. 1a und b a) Schallleitungsschwerhörigkeit Praktisch normale Knochenleitungskurve für Int. ne bei einem Luftleitungshörverlust von 50-60 db. Das Knochenleitungssprachaudiogramm zeigt gleiche Werte fast normale Knochenleitung bei einem Hörverlust von 50-60 db im Luftleitungs-sprachaudiogramm. b) Innenohrschwerhörigkeit Luft- und Knochenleitung decken sich praktisch bei Prüfung mit Intonen und Sprache.

kopfhörer 144 (Impedanz 2 Ω) und erhalten mit Normalhorenden die üblichen Bezugskurven. Da das Knochenleitungs-telefon 2/638 eine größere elektrische Leistung erfordert, koppeln wir es mit dem Lautsprecher ausgang (Impedanz 4 Ω) des Gerätes und ändern die Eichung so, dass bei Normalhorenden die Luft- und Knochenleitungskurven zur Deckung kommen. Die maximale Leistung des Gerätes betrug in Luftleitung 110 db, in Knochenleitung aber nur 90 db. Durch die Gummiumhüllung ist das Knochenleitungs-telefon so gedämpft, dass es keinen stärkeren Luftschall abstrahlt und wir tatsächlich nur knochenschall messen. Der eigentliche Messvorgang ist bekannt: in Abhängigkeit von der Intensität wird mit dem Zahlen- und dem Wortertest das prozentuale Zahlen- und Wortverständnis in Luft- und Knochenleitung gemessen. In Abb. 2 billen wir die mit Normalho-

Einton Audiogramm



Sprach Audiogramm



Von 5a und b MP 33 J Otosklerose a) Einton und Sprachaudiogramm vor der Operation. Das Knochenleitungssprachaudiogramm bestätigt die Diagnose und erlaubt die Voraussage über den zu erwartenden Hörgewinn. b) Postoperativer Gewinn nach Interposition eines für Eintöne und für Sprache.

renden ermittelten Luft- und Knochenleitungskurven für den Zahlen- und Worttest ab.

Nach Eichung des Sprachaudiometers und Ausmessung einer genügend grossen Anzahl Normalhorender gingen wir dazu über, auch Schwerhörige auszumessen. Um klare Ergebnisse zu erzielen und ein Überhören im Knochenleitung auszuschliessen bzw. eine Verübung zu vermeiden prüften wir zunächst das Hörvermögen einseitig tauber Schalleitungsgestörter bzw. immer das besser hörende Ohr unserer Probanden.

Bei einem einseitig Tauben mit Normalgehör auf der anderen Seite erzeugten wir durch Verschluss des Gehörganges eine künstliche Schalleitungsschwerhörigkeit und nahmen ausser der üblichen Luft- und Knochenleitungsschwellenkurve für Eintöne auch ein Sprachaudiogramm in Luft- und Knochenleitung auf. Wir fanden die in Abb. 3 abgebildeten Kurven.

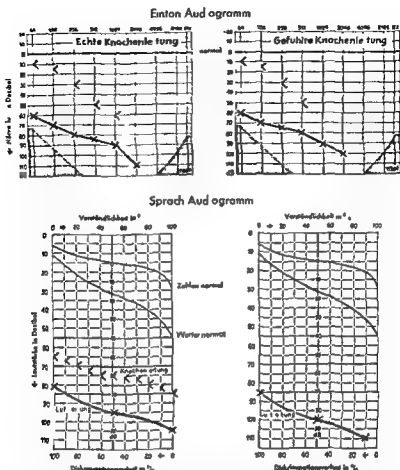


Abb 6 Diese Abbildung demonstriert den Wert der Knochenleitungssprachaudiometrie. Die beiden Eintonaudiogramme sehen praktisch gleich aus und doch handelt es sich in dem links abgebildeten Fall um eine kombinierte Schwerhörigkeit mit überschüssiger echter Knochenleitung, wie das zusätzliche Sprachaudiogramm in Knochenleitung deutlich zeigt. Im anderen Fall liegt aber eine reine Innenohrstörung vor, die überschüssige Knochenleitung für Eintöne war nur geföhlt.

Wir untersuchten eine grössere Anzahl reiner Schalleitungs- und Innenohrstörungen und fanden immer wieder ganz klare Ergebnisse. Wenn im Eintonaudiogramm bei grösserem Hörverlust in Luftleitung, eine überschüssige Knochenleitung gefunden wurde, so erhielten wir sprachaudiometrisch korrespondierende Werte und konnten so den schalleitungsbedingten Hörverlustanteil zusätzlich objektivieren. Deckten sich dagegen im Eintonaudiogramm Luft- und Knochenleitung, so war dies im Sprachaudiogramm ebenfalls der Fall. Die Abb. 4a und 4b bringen je ein typisches Beispiel dafür. Um insbesondere vor hörverbessernden Operationen ganz sichere Diagnosen stellen zu können, begnügen wir uns in der Zöllner'schen Klinik nicht mehr mit der bisherigen Eintonmessung in Luft- und Knochenleitung und

dem alleinigen Sprachaudiogramm in Luftleitung, sondern nehmen zusätzlich seit 1½ Jahren noch ein Sprachaudiogramm in Knochenleitung auf. Bei reinen Schalleitungsschwerhörigkeiten kann diese Vorsicht wohl überflüssig sein, nicht aber bei kombinierter oder hochgradiger Innenohrschwerhörigkeit. In Abb. 5 zeigen wir einen Fall mit typischer Otosklerose vor und nach Steigbügeloperation mit Interposition.

Waren die bisherigen Fälle völlig klar und brachten dem audiologisch geschulten Arzt nichts neues, so erscheint die letzte Abbildung problematisch. Bei Betrachtung der beiden Eintonaudiogramme glaubt man, dass in beiden Fällen eine kombinierte Schwerhörigkeit vorliegt. Das bisher übliche alleinige Luftleitungssprachaudiogramm gibt in derartigen Fällen uncharakteristische Kurven. Erst das zusätzliche Knochenleitungssprachaudiogramm zeigt, dass es sich in dem hier links abgebildeten Fall um eine kombinierte Schwerhörigkeit handelt, bei der eine Interposition vielleicht möglich wäre. Die überschüssende Knochenleitung im rechten Eintonaudiogramm war aber nur gefühlt. Luft- und Knochenleitungssprachaudiogramme decken sich, hier liegt eine reine Innenohrschwerhörigkeit vor, die nur mit einem Hörgerät versorgt werden kann.

ZUSAMMENFASSUNG

Bekanntlich geben Volltaube im Eintonaudiogramm sog. Fühlkurven an. Bei Fällen mit hochgradiger, fraglich kombinierter Schwerhörigkeit ist oft nicht genau zu entscheiden, ob die überschüssende Knochenleitung echt oder gefühlt ist. Vertäubungsmessungen, Nachsingen des Prüftones usw. klären das Bild ebenso wenig wie sprachaudiometrische Kurven, die bislang nur in Luftleitung aufgenommen wurden. Der Otorhinochirurg steht dann vor der heiklen Frage, ob er einen hörverbessernden Eingriff durchführen soll oder nicht.

Die vorliegende Arbeit berichtet über einen neuen Messvorgang: die sprachaudiometrische Prüfung in Knochenleitung. Die Grundlagen dazu wurden im Rahmen einer Dissertation erarbeitet und sind so weit abgeschlossen, dass diese Prüfung in die klinische Routinemessung eingebaut werden konnte. In der Freiburger Klinik prüfen wir unklare Fälle immer mit Eintonen und Sprache jeweils in Luft- und Knochenleitung. Gegenüber der „Ja-Nein-Aussage“ bei der Eintonaudiometrie, die — wie oben gesagt — ein Hörfehlen nicht ausschließt, erlaubt die Sprachaudiometrie in Knochenleitung, bei der Zahlen und Wörter nachgesprochen werden müssen, eine zusätzliche objektive Aussage. Bei fraglicher symmetrischer Schwerhörigkeit sollte dasjenige Ohr geprüft werden, in das beim Weber'schen Versuch lateralisiert wird, bzw. muss das Gegenohr vertäubt werden.

Wenn sich bei einer hochgradigen Schwerhörigkeit im Eintonenschwellenaudiogramm eine fraglich überschüssende Knochenleitungskurve zeigt, so war die „gute“ Knochenleitung im Eintonaudiogramm gefühlt und es handelt sich um eine Innenohrschwerhörigkeit. Sobald aber überschüssende sprachaudiometrische Knochenleitungskurven gemessen werden, so geben diese recht genau den schalleitungsbedingten Hörverlust an und ermöglichen damit eine bessere Indikationsstellung für hörverbessernde Operationen und eine genauere Prognose über den zu erwartenden postoperativen Hörerfolg als die bisher üblichen Messverfahren.

SUMMARY

In pure tone audiometry we can get so called "feeling curves" from totally deaf people. In hard of hearing patients with a questionable combined high degree of deafness, it is often impossible to discern whether bone conduction is true or felt. Measurements with masking do not lead to a clear diagnosis, either. In such uncertain cases, we write speech audiograms for air and bone conduction as well. Contrary to the yes/no statement in pure tone audiometry, speech audiometry in air and bone conduction definitely gives more objective results. There is a pure inner ear deafness when the two curves coincide. The exceeding bone conduction curve of a speech audiogram shows exactly the real part of the conductive hearing loss and enables us to get a better indication for operations in the middle ear.

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ÜBER EIN NEUARTIGES AUTOMATISCH GESTEUERTES ELEKTRONISCHES SPRECHGERÄT FÜR LARYNGEKTOMIERTEN¹

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Nach der Totalexstirpation des Larynx wegen maligner Geschwulstbildungen, welche in den letzten Jahren allgemein ansteigen, besteht für einen Grossteil der Patienten die Möglichkeit, die Oesophagussprache zu erlernen und damit wieder eine, wenn auch mangelhafte Verständigung mit der Umgebung zu erreichen. Jedoch gibt es immer wieder Fälle, welche die Speiseröhrensprache nicht erlernen und später genügend beherrschen können. Diese Gruppe ist nicht so klein; die Zahlen schwanken zwischen 10 % (Imre) und 33 % aller Laryngektomierten (Heaver, White & Goldstein). Für diese Patienten wäre ein technisches Hilfsmittel von aktuellem Interesse.

Angeregt durch die Leistungen der Elektronenindustrie, glaubten wir, dass es beim heutigen Stand der Technik möglich sein musste, ein Gerät zu entwerfen und mittels Transistoren und Miniaturelementen zu bauen, das die natürlichen Funktionen des operativ entfernten Kehlkopfes als Klangbildungsapparat in hohem Masse übernehmen konnte.

Es hat bereits bisher einzelne technische Vorschläge und Konstruktionen gegeben, von denen wir hier in Europa, z. B. die Pfeife von Tichioni erwähnen mochten. Aus den U.S.A. wollen wir den sogenannten „Floten Typ“ eines künstlichen Kehlkopfes anführen, der von der Grossfirma Western Electric gebaut und der Fa. Bell seit 1930 vertrieben wurde. Von diesem Typ mit der technischen Bezeichnung 2A (Abb. 1) wurden in den Staaten seither rund 5 500 Stück verkauft mit einer annähernden Konstante von 300 Stück pro Jahr, also ca. täglich 1 Stück.

1956 hat sich im National Hospital for Speech Disorders in New York ein Spezialkomitee für Fragen des künstlichen Kehlkopfes (Advisory committee on artificial larynxes) gebildet. In Zusammenarbeit mit diesem Komitee haben Western Electric und Bell ihr neuestes Modell entwickelt (Abb. 2). Diese Type, welche von aussen an den Hals gehalten wird und auch jeweils mit der Hand ein- und ausgeschaltet werden muss, soll im heurigen Herbst ausgeliefert werden.

Schon aus psychologischen Gründen ist zu fordern, dass ein solcher Apparat möglichst unauffällig sein soll. Der Kehlkopflose will nicht die

¹ Vorgetragen am Österreichischen Oto-Laryngologen Kongress in Zell am See (Salzburg) am 16. X. 1960.

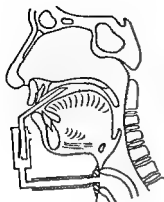


Fig. 1



Fig. 2

Abb. 1 Schema eines künstlichen Kehlkopfes vom sog. Floten Typ (nach Barney und Mitarbeiter)

Abb. 2 Neuester amerikanischer künstlicher Kehlkopf, der beim Sprechen aussen an den Hals gehalten wird und mit den Daumen jeweils ein- und ausgeschaltet werden muss (nach Barney und Mitarbeiter)

Aufmerksamkeit und das Mitleid der Umwelt erzeugen. Je besser es ihm gelingt seinen Defekt zu verbergen, umso leichter wird seine Resozialisierung sein. In Gerät, das den gegebenen physiologischen Gegebenheiten vollkommen Rechnung trägt, sollte aber auch ohne ständige Handbedingung auskommen können, was bisher bei keinem Apparat der Fall war. Gelingt aber eine automatische Selbststeuerung, dann wird der Patient beide Hände frei haben für jedwede andere Fähigkeit z. B. seines Berufes.

Vergleichen wir einmal die anatomischen und funktionellen Verhältnisse 1) bei einem Normalen und 2) bei einem Laryngektomierten und leiten daraus unsere Überlegungen ab.

Zur Anatomie

Ad 1) Atem- und Speisewege kreuzen sich und ad 2) Atem- und Speisewege sind getrennt. Der natürliche Klangbildungsapparat ist operativ entfernt.

Zur Funktion

1. Bei Analyse der Phonation ist festzustellen: Das Sprechen wird in der frühen Kindheit erlernt. Schon beim Kleinkind werden die für die Phonation erforderlichen Atemreflexe ausgebildet. Es wird nur mit Expirationsluft gesprochen. Der Atemstrom als mechanisch treibende Kraft bläst die beim phonatorischen Glottisschluss elastisch gespannten Stimmlippen mit einem gewissen Atemdruck an.

Nach dem 3. Gesetz über die Kompensation der Kräfte am menschlichen

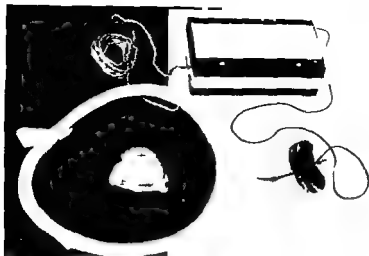


Abb 3 Einzelteile des künstlichen Kehlkopfes nach Dr. Herbert Pichler

Kehlkopf von Johannes Müller verhält sich die Stimmstärke dem Atemdruck gerade proportional

Nach der Laryngektomie liegen geänderte Verhältnisse vor. Auf dem Atemwege gibt es keine verstellbare natürliche Barriere mehr, es ist kein Glottisschluss möglich, denn Stimmglocke und Taschenbänder sind mit dem Kehlkopf entfernt worden. Bei der Phonation verpufft der normale Anströmungsdruck durch das Tracheostoma ins Freie. Wenn der Tracheektomisierte sprechen will, bläst er seinem gegenüberstehenden Gesprächspartner die Redeluft ins Gesicht.



Fig 4



Fig 5

Abb 4 Schema der Benützung

Abb 5 Der völlig unauffällig zu tragende ohne Halbleitung arbeitende künstliche Kehlkopf am Patienten

Es gehört nun zum Wesen des von uns entwickelten neuen Larynx, dass dieser Sprech Atemdruck zur Steuerung des Gerätes verwendet wird oder mit anderen Worten um den Betrieb des künstlichen Klangbildungsapparates vom Sprechwillen des Benutzers abhängig zu machen werden die synchronen Atemreflexe verwendet.

2 Beim Laryngektomierten sind Atemweg (Trachea) und Ansatzrohr getrennt. Der natürliche Klangbildungsapparat ist entfernt. Die Atemluft bewegt nicht mehr als arbeitender Luftstrom die Stimmblätter. Der Strom der Luft und damit der Strom des Schalles zum Mund und zu den Haupt artikulationsstellen (Gaumen Zunge Lippen) ist unterbrochen und wird vorzeitig für die Phonation ungenutzt nach aussen geleitet. Das Ansatzrohr und der Hypopharynx sind nunmehr eine fast ausschliessliche Schlundrohre geworden.

Als zweite funktionelle Forderung ist daher zu erheben: Platzierung der Schallgebung (des neuen künstlichen Klangbildungsapparates) möglichst wirksam im Ansatzrohr.

Nach Prüfung aller vorhandenen Möglichkeiten haben wir eine Einrichtung zur Mundschallerzeugung entworfen, welche darin besteht, einen kleinst lautsprecher in einer Oberkieferprothese unterzubringen.

Da die Sprachlaute im Ansatzrohr gebildet werden, jedem Vokal ein nach Form und Grösse vorbestimmter Resonanzraum entspricht, das Ansatzrohr bei Laryngektomierten aber im wesentlichen erhalten ist, wird ein im Artikulationsgebiet erzeugter Schall durch die psychomotorischen Artikulationsbewegungen eine genügende Modulation erfahren.

Durch diese zweite Forderung wird erreicht: Die beim Laryngektomierten bestehende mechanische Trennung zwischen Atemweg und Ansatzrohr wird auf elektrischem Wege übersprungen und die normale funktionelle Einheit des Sprachmechanismus dadurch wiederhergestellt.

Die technische Realisierung

Die Realisation des Projektes nach der Patentschrift wurde von Ingenieuren der I. a. Siemens und Halske, Wien durchgeführt. Die Steuerungskontrolle von der I. a. I. Reimer angefertigt. In den zahnärztlichen Belangen wurden wir von Doz. Dr. R. Wachter beraten und unterstützt. In dessen Ordination auch die Prothesenarbeiten erfolgten. Für die drahtlose Übertragung des erforderlichen Frequenzspektrums waren zahlreiche technische Versuche nötig, welche im R. I. a. 2 der I. a. Siemens stattfinden¹.

Das entwickelte erste Modell, über dessen Konstruktion erstmals am VI. Internationalen Thoraxkongress in Wien berichtet wurde, besteht aus 3 Hauptteilen:

¹ Aus Praktikationsgründen sei festgehalten: dass wir am 7. VIII. 1931 in Wien eine elektrische Kehlkopfprothese zum Patent angemeldet haben. Am 17. XI. 1938 wurde von uns diese Patent schriftlich durch die neue Patentanmeldung eines modifizierten und verbesserten auf national gesteuerten Gerätes ersetzt.

1. In einem Mikrosender mit Senderschleife welche unter der Kleidung um den Hals getragen wird

2. Einer Sprichkontaktdose welche als Impulsgeber in die Seele der Trachealkanüle eingebracht ist

3. In einem Kleinstlautsprecher, der als Impfungerteil in einer Oberkieferzahnprothese untergebracht ist

Abb. 3 zeigt Einzelteile des Gerätes und Abb. 4 und 5 den Patienten mit betriebsbereitem Gerät

Bisherige Erfahrungen am Patienten

Bei der Konstruktion des Gerätes und seiner Erprobung am Patienten sind sehr viele grosse und kleine Probleme aufgetaucht. Schwierigkeiten physikalischer, technischer, medizinischer und psychologischer Natur. Die bisherigen Ergebnisse seien in kurze zusammengefasst.

Das Gerät ist auf eine mittlere Sprechstimmlage von 150 Hz eingestellt. Die Qualität der Obertöne wird aber noch zu verbessern sein. Die Vokale A und O kommen gut, U weniger gut, die Vokale I und I schlecht zur Wahrnehmung. Nach Gewöhnung an die neue Zahnprothese ist die Artikulation der Konsonanten befriedigend besser als bei jenen Typen (wie z. B. Prototyp Tichionischer Pfeife) bei welchen die Lippen und Zähne nicht völlig geschlossen werden können. Die individuell auf den Atemdruck einstellbare Steuerung in der Trachealkanüle hat die theoretischen Erwartungen erfüllt und unsere physiologische Beweisführung bestätigt. Bei guter Einstellung erfolgt nach den einzelnen Worten und Sätzen automatisch die erforderliche Pause, was technisch einen Stromsparenden intermittierenden Betrieb bedeutet. Als Nachteil kann es bei grosser Nervosität des Patienten, starker Expiration, Hustenstossen etc. zu einem unangenehmen Jittern der Kontaktmembranen kommen.

Die Unterbringung des Lautsprechers und seiner Zusatzteile in der Oberkieferprothese war das Verdienst Doz. Dr. Wichter's. Allerdings ist die dauerhafte Speichelabdichtung des Lautsprecher-Systems schwierig und noch nicht endgültig gelöst. Die grosse Hauptschwierigkeit besteht darin, dass die Lautstärke bei drahtloser Übertragung von der Senderschleife aus noch nicht befriedigend ist. Auf die ebenfalls im Patent erwähnte Übertragung mittels Munddraht haben wir aus kosmetischen und medizinischen (starke Speichelabsonderung) Gründen bisher verzichtet, obwohl dann die nötige Lautstärke sofort erreichbar wäre. Zahlreiche Laborversuche zeigten, dass bei drahtloser Übertragung der Wirkungsgrad der Energieübertragung bisher leider sehr gering ist. Er beträgt weniger als 1% der aufgewandeten Energie. Es wurde das Gerät daher laufend verstärkt von ursprünglich 6 V auf später 12 V und jetzt 24 V Spannung. Hier ist noch rein technische Grundlagenforschung nötig. Weiter erforderlich werden spektralanalytische Kontrolluntersuchungen sein zur Ermittlung und Verfeinerung eines für alle Vokale gleich guten Frequenzspektrums. Untersuchungen, welche wir in

Zusammenarbeit mit der Wiener Technischen Hochschule planen abschliessend mochten wir feststellen, dass wir uns als wir dieses Gerät zum Patent angemeldet hatten natürlich bewusst waren dass bis zu einer serienmassigen Herstellung desselben noch eine Reihe von Verbesserungen notwendig sein werden

ZUSAMMENFASSUNG

Zehn bis 33% der Laryngektomierten erlernen nicht oder nur ungenugend die Oesophagussprache Ein modernes technisches Hilfsmittel ist daher von aktuellem Interesse Nach Erwähnung bisheriger Typen von künstlichen Kehlköpfen wird ein auf völlig neuen Konstruktionsprinzipien beruhendes automatisch gesteuertes elektronisches Sprechgerät beschrieben und demonstriert, das in den letzten 4 Jahren in Zusammenarbeit der I Universitätsklinik für Ohren, Nasen und Kehlkopfkrankheiten in Wien mit Firmen der österreichischen Elektronenindustrie entwickelt wurde Das Gerät besteht aus einem Mikrosender mit Senderschleife, einer sogenannten „Sprech Kontaktdose“ und einem kleinstlautsprecher, der in einer Oberkieferzahnprothese untergebracht ist und auf drahtlosem Wege versorgt wird Ziel der Zusammenarbeit war, keinen blossen Stimmersatz zu schaffen, sondern eine möglichst weitgehende Wiederherstellung der Sprechfunktion zu erreichen Hierzu waren vergleichende anatomische und physiologische Untersuchungen bei normalen und laryngektomierten Personen vorausgegangen, welche die Möglichkeit aufzeigten, die Sprech Atemreflexe zur Steuerung einer künstlichen Vokaltongebung zu verwenden Die nachfolgenden Experimente am Patienten haben die Realisierbarkeit dieses Erfindungsgedankens bestätigt Der Hauptvorteil des neuen künstlichen Kehlkopfes besteht darin, dass er völlig unauffällig zu tragen ist, ohne Handbedienung arbeitet, den normalen intermittierenden Sprechrhythmus Rechnung trägt und eine ungestörte Aussprache der Konsonanten gestattet Die bisherige Erprobung ergab, dass bis zur serienmassigen Herstellung noch einige technische Verbesserungen notwendig sein werden

SUMMARY

Between 10 and 33% of persons who have had their voice boxes removed in surgery cannot master oesophagus speech or master it sufficiently A modern technical aid is therefore of great interest After mentioning the various types of artificial larynxes used in the past a description and demonstration is given of an automatically controlled electronic speech aid which operates on completely new constructional principles and which has been developed during the past four years by the 1st University Ear Nose and Throat Clinic Vienna in cooperation with Austrian electronic industrial firms The aid consists of a microtransmitter with transmitter loop a so called speech contact plug and an extremely small loudspeaker which is housed in an artificial denture in the upper jaw operating without the use of wires The aim of the combined research was not just to produce a substitute for the voice but to create an aid which would restore the function of speech as far as possible This research followed a series of anatomical and physiological tests to study the differences between normal persons and persons who had undergone laryngectomy which pointed to the possibility of using the speech respiratory reflexes to control artificial vocal production The subsequent experiments with patients have con-

firmed the practicability of this train of thought. The main advantages of the new artificial larynx lies in the fact that it can be used without being seen and without manual operation, that it takes normal intermittent speech rhythm into consideration and that it allows undistorted pronunciation of the consonants. The tests carried out to date show that a few further technical improvements are necessary before the apparatus can be produced on a wide scale.

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A STUDY ON PENDULAR NYSTAGMUS

Pendular Nystagmus Its Contribution to the Understanding of Nystagmus Mechanisms

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The author studied the pendular type of nystagmus by using electronystagmography (ENG). Some of the findings obtained from the study appear to be common to nystagmus in general, i.e. not restricted to the pendular type. A comparison of the pendular and other types of nystagmus is therefore, highly interesting and beneficial.

Observations and discussions were made on (1) the difference of individual nystagmic patterns (2) the effect of opening or closing the eyelids on nystagmus, and (3) the factors which make pendular nystagmus jerky such as extraocular muscular vestibular central and optokinetic imbalances. Varied grades of nystagmus sensation dissociation (NSD) were found to exist in pendular nystagmus cases when vestibular and optokinetic stimuli were applied. The author is of the opinion that NSD in addition to directional difference observed in optokinetic after nystagmus is an important clue to nystagmus mechanisms.

The advent of electronystagmograph has made possible exact recordings of nystagmic patterns under various conditions. Many investigators in this field both here and abroad are enthusiastically taking a new look at nystagmic patterns electronystagmographically. The author studied pendular nystagmus with an expectation of valuable information being obtained about nystagmus in general. In pendular nystagmus ocular movements occur in an exaggerated way making a convenient pattern for observation and recording. Reactions as expounded in (2) and (3) paragraph 2 of the Summary, are specific types. Thus we are able to make comparative studies to deduce fundamental characteristics common to all types of nystagmus. Otolaryngologists since Barany have had a deep interest in utilizing vestibular nystagmus in clinical examinations. It is most important therefore to deepen the understanding of the intrinsic nature of nystagmus in order to minimize errors in evaluation of clinical tests utilizing nystagmus. A detailed study of each item briefly referred to in this paper will be shown in subsequent papers.

METHOD

Eye movements were recorded by a photo recording electronystagmograph (ENr). The time constant was 2 sec. for recording approximate eye

deviation and 0.05 sec for approximate eye speed. Only the horizontal component of ocular movements were recorded. Photographic recording was preferred to pen writing, because of the former's high fidelity.

Movement of the eyeballs was observed and recorded under the following optic conditions: with the eyes open and closed, covered behind lenses or Bartsch spectacles, gazing laterally, near and far accommodation, etc.

The influence of optokinetic labyrinthine or caloric and post-rotatory stimuli on spontaneous nystagmus was examined. Optokinetic stimulus was given by an electrically controlled rotating drum having a diameter of 1.8 m with vertical stripes inside. The electrically driven rotating chamber used for rotatory stimulation was mentioned in my previous paper.

OBSERVATIONS AND DISCUSSIONS

1. *There are as many different types of patterns of nystagmus as there are patients*

Fig. 1 illustrates examples of the horizontal nystagmic pattern. As illustrated, pendular nystagmus is not so typically pendular as would be expected from observations with the naked eye.

It was also observed in many cases that concentration or diversion of attention or changes in psychic condition modifies ocular movements and consequently nystagmus too. Some patients complained that general strain or fatigue precipitated spontaneous nystagmus. This complaint was proved to be justified. Other patients observed that maintenance of visual fixation was possible only for the initial several seconds and that the effort of fixing one's eyes on a point induced much brisker nystagmus resembling intention tremor. Fig. 2 shows a record of seizure-like nystagmus discharge occurring at intervals of every few seconds.

Our daily clinical experience shows that the patterns of induced jerky nystagmus even in normals are varied. Induced nystagmus in normals changes its appearance according to the external or internal condition of the individual in the same way as spontaneous pendular nystagmus does. In the future, an analysis of electronystagmogram will make it possible to utilize ENG to verify a patient's condition.

2. *Opening the eyelids usually enhances nystagmic movements greatly while closing the eyelids has an opposite effect*

Nystagmus with the eyes open differs quantitatively and sometimes even qualitatively from that recorded with the eyes closed, depending on the individual case (Fig. 3). Thus the nystagmus that appears with the eyes open is generally much more frequent and regular when compared to that observed with the eyes closed. There is sometimes a qualitative difference. Nystagmus appearing with the eyes closed is of a typical jerky variety, while with the eyes open it is a typical pendular type. In cases of pendular nystagmus to

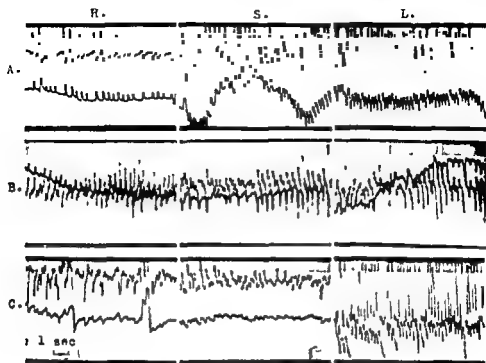


FIG. 1. Examples of pendular nystagmus (1, 2, 3) R., Lateral gazing to the right S., looking straight forward, L., lateral gazing to the left. Two curves in the illustrations are each recorded with a time constant of 2.0 sec. and 0.05 sec. Upward deviation of curves in each illustration represents ocular movements to the left downward to the right.

open the eyes in the dark has almost the same effect on nystagmus as to open them in the light. Therefore, it will be concluded that the effect of opening the eyes is not that of actual fixation but that of merely opening the eyes.

As many authors have pointed out, elimination of fixation is favourable for recording "true" vestibular nystagmus. However, elimination of fixation by closing the eyes is not always favourable, probably because closing the eyes exerts a suppressive influence on nystagmus, as demonstrated in pendular

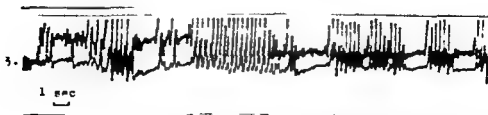


FIG. 2. Seizure-like nystagmus: alternation of periods of depression and facilitation of nystagmus at intervals of several seconds.

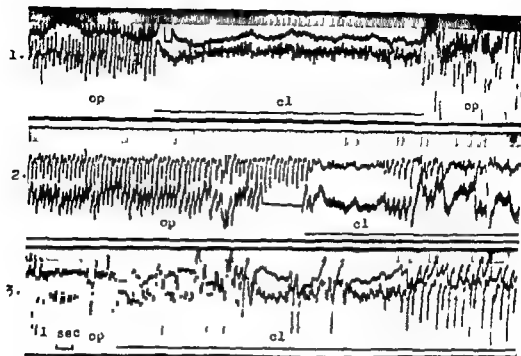


FIG. 3 The effects of closing of the eyes on nystagmus (op opened cl closed) 1 Complete suppression of nystagmus 2 nystagmus appears with a latent period of a few seconds after closing of the eyes 3 comparison of frequent and regular nystagmus with the eyes open and infrequent irregular nystagmus with the eyes closed

nystagmus cases. This suppressive influence on vestibular nystagmus when closing the eyes however appears variable individually from the slight to the extreme. True vestibular nystagmus appears most distinctly with the eyes open in the dark in some while in others most clearly with the eyes closed.

Vestibular nystagmus, which is jerky in type consists of two phases the slow and the quick. Activation of vestibular nystagmus is to be expected by facilitation of either phase. Both components change their appearance according to the degree of fixation though it is believed that the slow phase of nystagmus originates from the vestibular system while the quick one is a corrective motion of deviated ocular position promoted by the central neural mechanisms. Activation of the quick component is therefore probably achieved through activation of the central neural mechanisms especially that of the oculomotor system. In this connection opening the eyes must provide a favourable condition for vestibular nystagmus too. It is therefore comprehensible that opening the eyelids in the dark provides an ideal condition in most cases (Fig. 4).

Activation of pendular nystagmus by opening the eyelids is possibly caused by the will to look at something when the eyelids are opened. The electroencephalogram shows that opening the eyelids causes diffuse elevation of

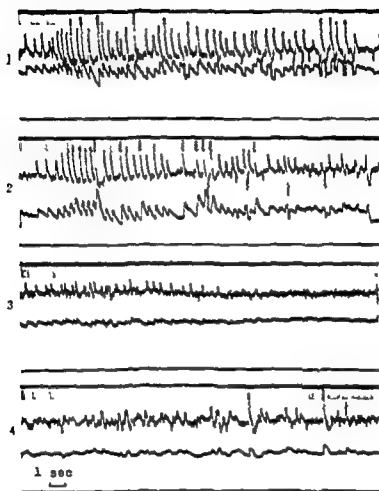


FIG. 4 Post rotatory nystagmus in a normal test subject. 1 Behind lens spectacles with the eyes open in a dark room. 2 Behind lens spectacles with the eyes closed. 3 Gazing at the point 100 cm off the eyes. 4 With the eyes closed.

cortical activity. This diffuse cerebral activation observed in EEG is presumed to show a generalized elevation of the activity level of the central nervous system including the brain stem, which has close connection with ocular movements.

It was observed that a typical jerks type of nystagmus appeared in pendular nystagmus cases when the eyes were closed, though difficult to explain, it is full of suggestion. It is conjectured that a new tonic imbalance may be formed in the central nervous system by closing the eyes, and jerks nystagmus appears instead of pendular because of suppression of the latter by closing the eyelids.

3 *Pendular nystagmus is changed into the jerky type with extreme ease*

A Tonic imbalance of the extraocular muscles A lateral gaze transforms most of pendular nystagmus into the jerky type with the quick phase to the side of the gaze (Fig. 1). This jerky nystagmus obtained by transformation of the pendular type differs from ordinary jerky nystagmus in its pattern as well as in its frequency as illustrated in the figure (compare this with the jerky nystagmus of Fig. 4). Congenital idiopathic nystagmus which usually has an asymmetrical neutral point is jerky in type away from the neutral point position.

In normals a certain feed back mechanism must be functioning to fix the eyeballs at a lateral position thereby resisting more or less the amount of unbalanced tonus between the extraocular antagonists. Thus the eyeballs are enabled to be fixed laterally and man can continue to look at a point located to the side. When the mechanism is disturbed or damaged jerky nystagmus appears. Continuance of extreme lateral gazing induces jerky nystagmus even in normals because of excess disparity of balance among the extraocular muscles and because of muscular fatigue. Paralysis of extraocular muscles is also responsible for the appearance of lateral gaze nystagmus. The characteristic lateral gaze nystagmus appearing in central neural diseases such as tumours occupying the posterior cranial fossa must be caused by central lesions of the mechanism. Lateral gaze nystagmus in pendular nystagmus cases must be caused by a disturbance of the fixation mechanism which is probably the very cause of pendular nystagmus itself.

B Vestibular imbalance Rotatory or caloric stimulation changes pendular nystagmus into the jerky type. Induced nystagmus in the cases under review however did not appear to be an indicator of the degree of excitement or sensitivity of the peripheral labyrinth because induced nystagmus with the eyes open is too active and also the duration of induced nystagmus is difficult to determine. In nystagmus reaction with the eyes closed the following variations were present:

(a) No detectable reaction

(b) Suppressed reaction varied individually from slight to extreme. Typical jerky nystagmus appeared in a few cases. Therefore nystagmic reaction induced with the eyes closed cannot be used as an indicator of vestibular excitability.

Vestibular nystagmic reactions rotatory or caloric as is widely known show a large individual variation even in normals. The difference between nystagmic reaction induced with the eyes open and that with the eyes closed is also large in normals. A highly uncertain nystagmic reaction in pendular nystagmus cases against vestibular stimuli as observed in this study suggests a difficulty of utilizing vestibular nystagmic reaction in clinical tests in cases other than in pendular nystagmus. It appears to be within the range of possibility that vestibular nystagmic reaction induced in apparent normals may

show the same kind and even the same amount of variety as in pendular nystagmus cases

C Central imbalance In alternating nystagmus nystagmus alternates its direction spontaneously from the right to the left at fairly constant intervals of c 120 sec. It was observed that changes in psychic conditions affected the period of alternation as well as external stimuli including that applied to the vestibular labyrinth.

Directional preponderance of nystagmic response which appears in brain tumors or other disorders in the CNS is regarded as a manifestation of imbalance. The second and third phases of induced vestibular nystagmus are considered to be caused by alternating compensatory tonic changes in the CNS. There is therefore the question of alternating nystagmus being closely connected with the second and third phases of induced nystagmus.

Periodic alternating nystagmus such as was observed in the cases under review may be regarded also as a manifestation of alternating central imbalance appearing visibly as nystagmus due to abnormally activated ocular movements. As anticipated it was observed that the period of alternation was influenced though the influence was slight in these cases by external or internal stimulation. Is it possible to assume that in normals the existence of such periodical alternation of central imbalance does not appear visible as nystagmus?

II Optokinetic imbalance Observation on reactions of pendular nystagmus to horizontal optokinetic stimulation in cases under review is outlined as follows

(a) No reaction (optische Drehstarre)

(b) Positive reaction

(1) Spontaneous nystagmus is inhibited

(2) Spontaneous nystagmus is modified

(i) Discrimination between the slow and the quick phase of induced nystagmus is difficult

(ii) Discrimination is possible but the quick phase runs against rotating stripes (typical reaction). This differs from normals in that there is no parallel between the speed of the slow phase of induced nystagmus and the angular velocity of the rotating stripes.

(iii) Discrimination is possible but the quick phase runs towards rotating stripes (atypical reaction). There is also almost no parallel between the speed of the slow phase of nystagmus and the angular speed of the drum.

Optokinetic reaction to one direction does not always occur in the same type as that to an opposite direction (II, 1, 2). There were found almost every combination of the above mentioned types of reactions in the cases studied.

Optokinetic nystagmic reaction in normals is entirely different from that

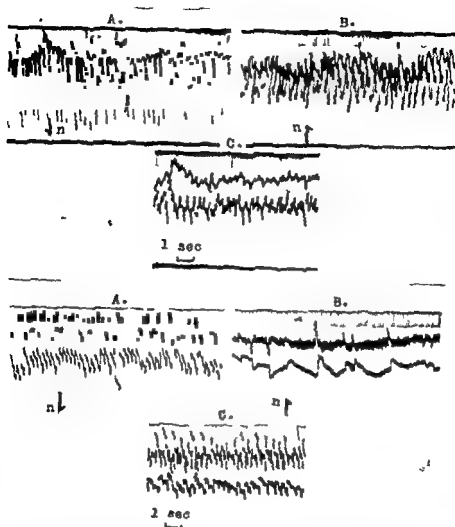


FIG. 5 Reaction against optokinetic stimulation in two cases of pendular nystagmus. A reaction to the moving stripes to the left side. B reaction to the moving stripes to the right side. C spontaneous nystagmus. n, direction of induced nystagmus in normal subjects.

observed in pathologic cases. In normals, optokinetic nystagmus is a typically jerky type of nystagmus and consists of both the slow and quick phases. During the slow phase the eyeball makes a follow movement with almost the same angular velocity as that of the rotating drum. With every quick movement the eyeball returns to its initial position. The author believes that optokinetic reactions will form a most important test for topographic diagnosis of diseases in the CNS though it is now used mainly for differential diagnosis of ocular nystagmus.

E. *After nystagmus following optokinetic stimulation—mobilization of nystagmic movements.* In several cases after nystagmus following optokinetic stimulation was observed, active and persistent. As reported by other investigators, the jerky type of nystagmus induced in pendular nystagmus cases

has a tendency to persist long after stimulation ended. There was observed a remarkable directional difference of after nystagmus reaction in some cases between right sided and left sided rotation while there was no detectable difference in others.

Thus optokinetic stimulation mobilizes nystagmic movements and keeps for some time directional possibly in the centrum. Directional difference between right sided and left sided after nystagmus possibly shows that there is a directional difference in the CNS. The foregoing promises a possibility of optokinetic after nystagmus being used as a test for examining tonic imbalance in the CNS.

4. Nystagmus-sensation dissociation

A sensation of dizziness which usually accompanies long duration optokinetic stimulation in normals was not complained of by the patients dealt with. Vertigo which also accompanies strong labyrinthine stimulation did not appear or was minimal in the majority of the cases studied. Even with strong caloric stimulation accompanying vertigo was slight or absent while vestibulo-ocular or vestibulo-spinal reaction was remarked. This was especially so with caloric stimulation. The author has designated this condition as nystagmus-sensation-dissociation (NSD) when the nystagmic reaction is extremely active and the vertigo is minimal.

Pendular nystagmus cases from the beginning have neither vertigo nor dizziness in spite of continuously existing active nystagmus thus it may be said they have NSD throughout their lives.

It is our usual experience to find during clinical examination that vertigo sensation accompanying induced nystagmus especially caloric nystagmus is different from case to case. Namely this difference may show that there is a varied grade of NSD in those cases other than pendular nystagmus. In central nystagmus such as is seen in multiple sclerosis or brain stem lesions NSD always exists. NSD is also possibly important in studying individual difference of liability to motion sickness and it may come to be used as examination media of the autonomic nervous system.

ZUSAMMENFASSUNG

Der Pendelnystagmus wurde mittels der Elektronystagmographie (LNG) studiert. Es ergab sich, dass dieser Typus des Nystagmus in bestimmten Punkten gemeinsam mit den anderen sind, d. h. sie sind nicht immer dem Pendelnystagmus eigen, weswegen der Vergleich der Erscheinungen des pendelschlägigen Typus mit denen der anderen Typen interessant und belehrend sind. Es wurden somit die folgenden Beobachtungen durchgeführt: 1) individuelle Unterschiede des Nystagmismusters; 2) Erfolg des Offens und Schliessens der Augenlider auf den Nystagmus; 3) Faktoren, die dem Pendelnystagmus eine rückschlägige Beschaffenheit geben wie z. B. die von den Aussenaugenmuskeln hervorgerufenen vestibulären, zentralen und optokinetischen Tonusdifferenzen. Bei der Anwendung der vestibulären und optokinetischen Reize für den Pendelnystagmus wurde die Nystagmus-Schwindel-Dissoziation

(NSD) verschiedenen Grades beobachtet. Es sei betont, dass die NSD sowie die Richtungsdifferenz, die im optokinetischen Nachnystagmus zu beobachten sind, zum Verständnis des Mechanismus des Nystagmus einen wichtigen Anhaltspunkt geben wird.

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CERUMINOMA IN THE TYMPANIC CAVITY

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A case of benign tumour in the tympanic cavity originating from the ceruminous glands is reported. The symptoms were like those of subacute otitis and the patient was first treated by atticotomy. Later on when the symptoms returned the diagnosis of ceruminoma was made on microscopical examination and a resection of the processus mastoideus and the tympanic cavity was performed. In the available literature 26 previous cases have been found among which one with localization to the tympanic cavity. The diagnosis, treatment and prognosis are discussed. Radical operative treatment and exact checking are recommended.

The so called ceruminous glands in the auditory meatus are a variation of the apocrine sweat glands. In conjunction with the sebaceous glands associated with the hairs they secrete cerumen which is thus made up of two components viz. the viscous sticky secretion of the sebaceous glands and the more fluid secretion from the ceruminous glands (Collins 1931). Accordingly we often find that the two types of glands have a common outlet in the hair follicles.

Most of the ceruminous glands are in the outer third of the auditory meatus corresponding to the hairy skin in the cartilaginous part but they may also be present in the deeper parts of the auditory meatus usually in the postero-superior wall.

On microscopical examination they are seen to be like the sweat glands tubular glands made up of a secretory and an excretory part. The secretory part consists of an outer myoepithelial layer and an inner layer of cuboidal cells or somewhat higher columnar cells with large round nuclei. In the cytoplasm near the lumen there are granules partly pigmented. On the surface there are protrusions of cytoplasm said to be characteristic of the apocrine glands. In the excretory part of the gland there is a two layered flattened epithelium the outer layer of which is assumed to be analogous to the myoepithelial layer of the secretory part. As a characteristic feature of ordinary sweat glands the tendency of the secretory as well as of the excretory part to ramify is emphasized. The myoepithelial element is said to be especially pronounced and the glands as a whole to assume considerably larger proportions.

From these glands, tumours may originate in rare cases. In the literature, such cases have been reported by Johnstone, Lennox & Watson (1937), who record 5 cases and review 10 previously described ones, including the earliest reported of all (Haug, 1894). Cases have also been described by Rutlin (1933), O'Neill & Parker (1957), Berlin (1949), July (1957) and Brannon & Fischer (1951).

In typical cases the tumour appears as a reddish or bluish polypous structure in the outer end of the auditory meatus. Histologically the structure is either an adenoma or an adenocarcinoma. The picture may vary considerably, and different types have actually been described on the basis of the microscopical findings (Johnstone *et al.*, 1937). One feature usually recurs, however, viz. the tendency here and there to form a two layered epithelium. The epithelial elements are found to be arranged as trabeculae, tubuli or as larger clusters with cystical cavities. The cells are eosinophilic cylindrical cells, medium sized spheroid cells and, occasionally, small darkly stained cells. The stroma is made up of finely reticulated, rather richly vascularized connective tissue, with larger or smaller infiltrations of lymphocytes and, occasionally, of plasma cells.

The symptoms in the pronounced cases arise mostly from the obturation of the auditory meatus, most often taking the form of deafness, discharge from the ear and pain, more rarely of tinnitus and, occasionally, facial palsy. Diagnosis will usually have to be made by biopsy, the differential diagnoses being mainly epithelial papillomata, pavement cell carcinomata and sialomata invading the auditory meatus.

Of the 26 cases reported in the available literature, 17 occurred in men, and 9 in women. Grouping by age was non-contributory, the youngest patient being 14 years old, and the oldest 78. As already mentioned, the usual site of the tumour is in the outer part of the auditory meatus, where most of the glands are. However, the tumours may also occur elsewhere, as will be seen in the case reported here.

Case Report

The patient was a 41 year old man who stated that he began to have bad hearing on the right side in 1951 but had no accompanying symptoms of any kind. He was then treated with paracentesis by a privately practising specialist, later with air douches and short waves, but this treatment had no noticeable effect.

Later on some tissue was removed from the tympanic membrane, and, finally, in 1955, a biopsy was made of the auditory meatus, followed by microscopical examination. The conclusion drawn after this examination was: adenoma like, presumably benign tumour tissue of sweat gland tumour structure, possibly originating from ceruminous glands.

The patient was then admitted to this department, where the findings on admission were as follows. All tympanic membrane injected, perhaps slightly swollen, with firmly adhering cerumen covering the central and postero superior part. Whispering voice 0.5/more than 3 metres. Rinne +/ Weber lateralized to the right.

BBB no nystagmus No facial palsy Audiometry right conductional loss of hearing of on an average 60 decibels Caloric test (44°C) normal

With no knowledge of the microscopical findings mentioned above the diagnosis of right subacute otitis media was made and right attico antrotomy \pm m Wullstein was performed During this operation a slightly irregular but otherwise normal cellular system was found The antrum was full of fleshy tissue extending into the attic and down into the middle ear Except for a negligible amount anteriorly most of the tissue was removed No defects of the tympanic membrane were in evidence The postoperative course was uncomplicated and the patient was discharged having a scanty secretion from his right ear Microscopical examination of the tissue removed from the attic showed small papillomatous fragments of tissue made up of somewhat vascularized stroma consisting of small cells and fine strands and of fibrous stroma The surfaces were covered by remnants of a cylindrical epithelium No conclusion could be drawn from these findings

The patient reported frequently for follow up examinations but apart from an insignificant secretion from the cavity presented no findings of special interest until the end of 1957 when a granulation like structure covering the tympanic membrane was found A biopsy specimen showed adenomatous structures of the sweat gland type with no signs of malignancy and the patient was again admitted to the department In January 1958 re operation of the right sided attico antrotomy was then performed with resection of the mastoid process and the tympanic cavity

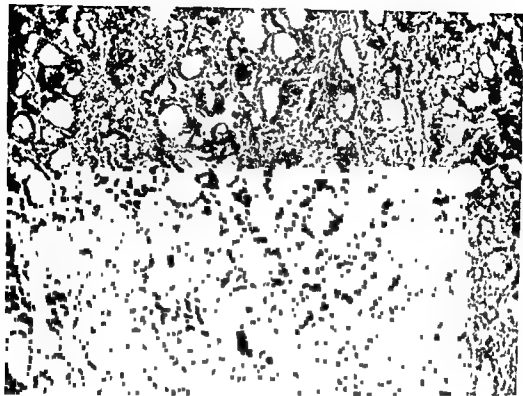
A narrow cavity was entered From the attic a large mass of granulation tissue projected into the tympanic cavity making the eardrum bulge The bridge was cut through and the malleus and incus were seen to be embedded in solid granulation masses that were bleeding copiously The granulation tissue was removed together with the ossicles of the ear the area around the stapes was left untouched however The postoperative course was uncomplicated

The patient has since then been repeatedly examined most recently at the end of August 1959 when some moisture in the cavity was found but no macroscopical signs of recurrence no dizziness and no pains

Microscopical examination of the tissue removed confirmed the diagnosis of adenomatous structures of the sweat gland type Pieces of tissue were found consisting of stroma fairly rich in cells with fine strands and somewhat vascularized surrounding densely arranged tubular somewhat sinuous structures composed of cylindrical low to medium high slightly eosinophilic epithelial cells containing some cytoplasm The nuclei which were rather rich in chromatin, were located in the basal part of the cells In the lumina a slightly eosinophilic secretion was present No mitoses were seen On several sections eosinophilic granules were seen in the cytoplasm of the cylindrical cells

Here we have then a case of ceruminous gland tumour in the tympanic cavity a site which is obviously rare but still one which has been described previously (Juby 1957) In the case reported by Berlin (1949) a tumour presenting the same microscopical picture was found at the porus acusticus internus situated extradurally and causing destruction of the bone with concomitant paresis of the 7th and 8th cranial nerves

Now what makes a tumour occur in the tympanic cavity in some cases cannot be definitely explained One may assume that the tumour has pene-

FIG 1 $\times 120$

trated the tympanic membrane, which is what has probably happened when parts of the tumour are encountered in the auditory meatus. In the case reported here, this is presumably the explanation, as the patient's statement that at a rather early juncture "some dead flesh" had been removed from his eardrum suggests some structure or other, deep in the meatus. The biopsy made in 1955, offering the earliest suggestion of the diagnosis, was also from the meatus.

Berlin's case of an intracranial ceruminous gland tumour shows, however, that this variety of tumour may arise outside the area of the auditory meatus, always provided, of course, that tissue derived from the ectoderm is present. The mucous membrane of the tympanic cavity contains no glands, to say nothing of apocrine sweat glands, but a tumour may conceivably have originated from an ectodermal vestige in contact with the middle ear. Considering the rarity of these tumours and moreover their extremely rare location in the middle ear, it is no wonder that the true nature of the patient's condition remained uncertain for a long time and was mistaken for inflammatory changes.

Differential diagnosis from otitis media chronica is consequently difficult and can, in theory, if otoscopic examination is not helpful, be made only if the cellular system of the mastoid process is found to be normal. Differential

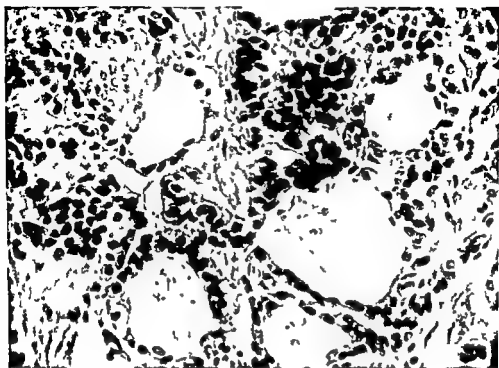


FIG. 2 6x0

diagnosis from other tumours in the middle ear whether carcinomata tumours of the glomus jugulare and so forth will be even more difficult not to say impossible unless a biopsy can be made. The vascularity of the ceruminous gland tumours is however somewhat lower than that of the last mentioned tumours.

Certain features of the clinical course of the case reported here agree well with the cases previously reported. The tendency to recurrence for example is well known and is best illustrated by the case reported by Johnstone, Lennox & Watson where the tumour recurred 10 times. This tendency is presumably associated with the difficulty of removing the tumour completely which applies both to the location in the auditory meatus and the localisation in the middle ear. Ceruminous gland tumours moreover demand interest because signs of malignancy will often be encountered.

In the 26 cases selected for comparison with the case history under review the microscopical diagnosis of adenocarcinoma is found in ten of them. Fatal outcome with regional glandular metastases was described in one of O'Neill & Parker's cases. There appears to be a tendency for the recurring tumours to assume ever increasing histological malignancy. Because of these two factors, recurrences and malignancy, one has to be most observant when treating a patient with a ceruminous gland tumour. The tumour must

be removed as completely as possible in the auditory meatus by deep excision into sound tissue and in the tympanic cavity by radical operation. The less radical procedures—superficial extirpation and atticotomy—will hardly achieve the desired object as already stated they are partly responsible for the frequent tendency to recurrence. Postoperative X-ray therapy may prove able to reduce the frequency of recurrence still more. It needs hardly be mentioned that because of the risk of recurrence the patients have to report for frequent follow up examinations for several years.

ZUSAMMENFASSUNG

Ein Fall von ceruminoma in der Paukenhöhle wird berichtet. Die Symptomen waren denen der subakuten Mittelohrentzündung ähnlich und der Patient wurde zuerst mit einem atticotomy behandelt.

Später wurde der Fall als ceruminoma diagnostiziert und eine Radikaloperation wurde unternommen.

Es sind früher von 26 Fällen in der Literatur berichtet worden darunter einem mit Lokalisation in der Paukenhöhle.

Diagnose, Behandlung und Prognose werden diskutiert. Radikale Entfernung und sorgfältige Überwachung empfehlen sich.

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REGISTRIFUNG VON INTRA AURALEN MUSKELREFLEXEN DURCH CUTANE REIZUNG BEI MENSCHEN

Vorläufige Mitteilung

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Die Haut des Gehorganges und des äusseren Ohres mit der nächsten Umgebung ist beim Menschen mit sensorischen Fasern des n. facialis n. trigeminus, n. vagus und m. glossopharyngeus ausgerüstet. Die Nervenfasern dieses Hautbezirkes enden bei Ratten im Nucleus tractus solitari. Von da aus können die Impulse wahrscheinlich durch Zellen der reticulären Substanz auf die motorischen Facialis und Trigeminuskern überführt werden.

Durch Luftstimulation des oben erwähnten Hautbezirkes haben wir eine klinisch einfach anwendbare Methode gefunden, mit der man beim Menschen nicht akustische intraaurale Muskelreflexe hervorrufen kann. Die Reflexbahnen sind wahrscheinlich die gleichen wie bei Ratten. Die vorläufigen Untersuchungen deuten darauf hin, dass der Stapediusreflex sowohl gekreuzt als ungekreuzt ist, während der Tensorreflex im wesentlichen ungekreuzt ist.

Akustische Muskelreflexe

Bei den meisten Versuchstieren (Katzen Hunden Kaninchen Meerschweinchen) rufen Töne von hoher Intensität eine doppelseitige reflektorische Kontraktion der intra auralen Muskeln — m. stapedius und m. tensor tympani hervor. Dieses ist durch eine Reihe Untersuchungen erwiesen (Crowe & Mitarb. (3) Kobrak (12) Wiggers (30) Okamoto & Mitarb. (23) Wever & Lawrence (29) Gisselsson & Mitarb. (6) Versall (28) Galambos & Rupert (6)).

Bei Affen ist es nicht gelungen, einen akustischen Tensorreflex nachzuweisen, nur Kontraktion des m. stapedius durch akustische Reizung (Kato (8)). Viele Forscher (Jepsen (7) v. Bekesy (1) Lüscher (17) Klockhoff & Andersen (11)) meinen, dass die Verhältnisse beim Menschen die gleichen sind wie bei Affen. Terkildsen (2) kam zu einem anderen Ergebnis. Ob es einen sicheren akustischen Tensorreflex beim Menschen gibt, ist also noch nicht eindeutig erwiesen.

Mit Hilfe von Messung der akustischen Impedanz des Trommelfells hat man eine Reihe Untersuchungen über die akustisch ausgelösten Muskelreflexe beim Menschen durchgeführt (Metz (19) Jepsen (7) Thomsen (26) Møller (22) Terkildsen (2)).

Nicht akustische Muskelreflexe

In einem Versuch an Katzen und Kaninchen hat Kato (8) gezeigt dass Schmerzreizung mit begleitender Abwehrbewegung eine reflektorische Kontraktion der intra auralen Muskeln hervorruft Ähnliche kann man auch durch relativ schwache mechanische Reize auslösen z B Berührung des äusseren Ohres und der unmittelbaren Umgebung oder der Haut des Gehörganges Durch intracraniale Durchschneidung des n facialis oder n vagus haben Kato (8) und Lorente de No (14) gezeigt dass die afferenten Impulse durch den auricularen Zweig des n vagus geleitet werden

Auch andre Reizarten z B Nadelstich in das ovale Fenster Durchschneidung des n trochlearis oder elektrische Reizung des n facialis können reflektorische Kontraktion der intra auralen Muskeln hervorrufen Schlag gegen die Zähne oder Schlucken scheinen ähnliche Wirkung zu haben

Mit nicht akustischen Muskelreflexen beim Menschen ist bis jetzt wenig gearbeitet worden wahrscheinlich weil diese Reflexe schwer hervorzurufen waren Während der Arbeit mit akustischen Muskelreflexen war Iepsen (7) nicht in der Lage nicht akustische Muskelreflexe zu beweisen die stören auf die Messresultate einwirkten Auch Iuscher (17) konnte nicht bei allen Menschen nicht akustische Muskelreflexe registrieren Pichler & Bornschein (24) und später Klockhoff & Anderson (10) haben auf etwas verschiedene Art gezeigt dass schwache elektrische Reizung der Haut des Gehörganges Kontraktion der intra auralen Muskeln hervorruft Durch akustische Impedanzregistrierung und elektro cutane Reizung sind Klockhoff & Anderson (10) zu dem Ergebnis gekommen dass es sich im Wesentlichen um eine Stapediuskontraktion handelt Die Impedanzvergrößerung ist phasisch d h sie kann nicht durch anhaltende Reizung auf gleicher Höhe gehalten werden und schwache Reize bewirken eine einseitige Impedanzänderung stärkere eine doppelseitige Eine akustische Impedanzänderung ist bekanntlich doppelseitig (Iuscher (16) Lindsay & Mitarb (13) und statisch d h sie kann mehrere Sekunden auf gleicher Höhe gehalten werden vorausgesetzt dass der Reizung anhält Es scheint also ein gewisser Unterschied zwischen der akustischen und der elektro cutanen Impedanzänderung zu bestehen

Ausserdem haben Klockhoff & Anderson (11) gezeigt dass ein flüchtiger und unerwarteter Luftstrom gegen eine der Orbitalregionen beim Menschen eine generelle Muskelkontraktion hervorruft Dabei kommt es zum Augenschluss zur Strammung der Halsmuskulatur und als Glied in dieser Abwehrreaktion auch zur Kontraktion des m tensor tympani und wahrscheinlich auch des m stapedius Der orbitale Tensorreflex ist phasisch kurz andauernd (0.5 sek.) und nimmt bei anhaltender Reizung an Stärke ab Die afferenten Bahnen für diesen Reflex sind nicht bekannt

Mangold & Eckstein (18) und Iuscher (16) haben gezeigt dass eine Kontraktion der intra auralen Muskeln auch bewusst hervorgerufen werden kann Viele Menschen können nämlich einen tiefen brummenden Ton hören wenn sie ihre periorbitale Muskulatur willkürlich kontrahieren

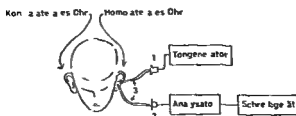


Fig. 1 Schematische Darstellung der Apparatur 1 Tongeber 2 Messmikrophon 3 Plastikschlange

Eigene Untersuchungen

Bei Arbeit mit akustischer Impedanzmessung haben wir eine sehr einfache, klinisch leicht anwendbare Methode entdeckt, beim Menschen intra-aurale Muskelreflexe hervorzurufen nämlich Blasen gegen das äussere Ohr und einen bestimmten Hautbezirk in der Umgebung des Ohres. Eine weitere Erforschung dieser Methode ist zur Zeit noch im Gange und eine ausführliche Beschreibung ist in Ausarbeitung. Der Zweck dieser vorläufigen Mitteilung ist daher nur eine kurze Beschreibung von Technik, Methode und möglicher Reflexbahnen zu geben.

Technik

Um die intra-auralen Muskelreflexe zu registrieren haben wir im Wesentlichen die von Klockhoff & Anderson (10) angegebene Apparatur benutzt, aber ohne elektrischen Stimulator. Man nimmt einen Sinuston (500 Hertz) aus dem Telefon eines gewöhnlichen Audiometers und leitet ihn durch eine Plastikschlange und weiter durch ein dünnes Polyethylenrohr gegen das Trommelfell. Ein Teil der Lautenergie wird resorbiert, aber ein Teil wird zurückgeworfen von einem anderen Polyethylenrohr aufgefangen und durch eine Plastikschlange zu einem Mikrophon geleitet. Das Mikrophon transformiert die Schallwellen in elektrische Schwingungen welche durch einen Verstärker zu einem Frequenzspektrometer geleitet werden. Wenn die Spannung von Trommelfell und Gehörknöchelchen konstant ist so wird auch die Energiemenge welche zurückgeworfen wird konstant sein und der Zeiger des Spektrometers steht still. Wenn sich die intra-auralen Muskeln kontrahieren wird das Trommelfell und die Kette der Gehörknöchelchen gespannt und die zurückgeworfene Energiemenge nimmt zu was sich in einem deutlichen Ausschlag im Spektrometer kundtut. Je stärker die Muskelkontraktion ist umso mehr Lautenergie wird vom Trommelfell zurückgeworfen und die Ausschläge auf dem Instrument werden grösser. Koppelt man ein Schreibgerät an das Frequenzspektrometer kann man die Impedanzänderungen auf einem Papierstreifen aufzeichnen (Fig. 1).

Methode

Unsere Ergebnisse sind in kurze folgende

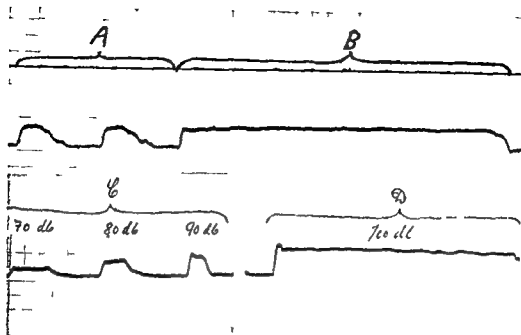


Fig 2 Impedanzänderung bei Luft und akustischer Stimulation des kontralateralen Ohres A, Unterbrochener Luftstrom B Anhaltender Luftstrom C, Unterbrochener akustischer Reiz von steigender Intensität (1000 Hertz) D Anhaltender akustischer Reiz (1000 Hertz — 100 db)

A Blasen gegen das kontralaterale Ohr

Richtet man einen schwachen Luftstrom gegen das äussere Ohr, z B Blasen mit einem Politzerballong, bekommt man eine tonische Kontraktion des m stapedius der gleichen und der entgegengesetzten Seite In geeigneten Fällen und durch Exploration des Mittelohres im Operationsmikroskop konnten wir unsere Ergebnisse verifizieren Die Kontraktion des m stapedius kann man lange Zeit allein durch einen schwachen Luftstrom auf gleicher Höhe halten Auf diese Art unterscheidet sich unser „Blasreflex“ von der phasischen Stapediuskontraktion die Klockhoff & Anderson (9) bei elektrischer Reizung der Haut des Gehorganges fanden Die Impedanzänderungen, die wir durch Blasen gegen das kontralaterale Ohr messen konnten entsprechen genau den Änderungen, die man durch akustische Reizung des gleichen Ohres erhält (Fig 2)

Wie früher erwähnt, meinen die meisten Forscher, dass es beim Menschen keinen akustischen Tensorreflex gibt Unsere Untersuchungen weisen im Grunde in die gleiche Richtung Nur bei einem einzigen Patient mit einseitiger Otosklerose war es vor der Operation möglich, auf diesem Ohr, durch akustische Reizung des anderen Ohres, wo das Gehör normal war, eine schwache Impedanzänderung hervorzurufen

Sowohl durch Blasen wie durch akustische Reizung scheint es sich also im Wesentlichen um eine Stapediuskontraktion zu handeln, die man durch Reizung des kontralateralen Ohres erhält Nur bei einigen Patienten, bei

denen die Sehne des m. stapedius durchschnitten war oder wo der ganze Stapes entfernt und durch Stahldraht und ein Fettstück ersetzt war (Schuknechts Operation) war es möglich durch Luftstimulation des kontralateralen Ohres kleine Impedanzänderungen zu registrieren. Dieses muss deshalb durch Reflexaktivität im m. tensor tympani verursacht sein.



FIG. 3 Darstellung des Hautbezirkes von dem aus die intra-auralen Muskelreflexe durch Luftstimulation ausgelöst werden

Das Hautgebiet von dem aus man mit Luftstimulation Kontraktion der intraauralen Muskeln hervorrufen kann ist ziemlich begrenzt und umfasst im Wesentlichen die Vorderseite der Ohrmuschel, das Ohrfläppchen und einen Hautbezirk, der sich 2–4 cm vor dem Tragus erstreckt (Fig. 3).

Das empfindlichste Gebiet ist die Hinterseite des Tragus und die Haut, die die Gehörgangsöffnung umgibt. Bei den meisten Menschen kann man von dem eben erwähnten Hautbezirk aus auch durch leichte Berührung mit einem Wattebausch eine Impedanzänderung hervorrufen, aber das ist bei weitem nicht so gut und sicher wie ein Luftstrom. Sogar wenn der Gehörgang sorgfältig tamponiert ist, kann man durch Blasen gegen das äussere Ohr eine Muskelkontraktion hervorrufen, aber diese ist dann schwächer, wahrscheinlich weil die akustische Komponente des Luftstromes bedeutend reduziert ist. Sogar bei Ohren, die durch einen Cochleaschaden taub sind, kann man durch Luftstimulation Impedanzänderung der entgegengesetzten Seite hervorrufen, sofern bei diesem Ohre keine mechanische Komponente vorliegt. Es kann also kein akustischer Reiz sein, der den Reflex hervorruft.

II. Blasen gegen das homolaterale Ohr

Bläst man gegen das Ohr, auf dem die Impedanzänderung registriert wird (homolaterales Ohr), erhält man Kurven, die etwas unterschiedlich sind von denen, die durch Blasen und akustische Reizung des kontralateralen Ohres entstehen. (Vergleiche Fig. 2 und Fig. 4).

Es scheint als ob zu der Impedanzverstärkung, welche durch Kontraktion des m. stapedius hervorgerufen wird, zusätzlich eine Reihe kleiner phasischer Ausschläge kommen, die man möglicherweise als Tensoraktivität bei dieser Form von Reizung erklären kann. Das deutet darauf hin, dass der

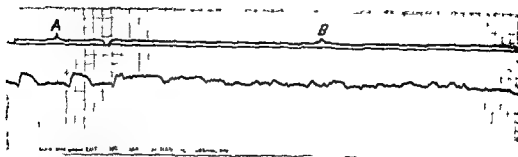


Fig 4 Impedanzänderung durch Luftstimulation des homolateralen Ohres A Unterbrochener Luftstrom B, Anhaltender Luftstrom

Tensorreflex im Wesentlichen ungekreuzt ist, der Stapediusreflex sowohl gekreuzt als ungekreuzt

Bei Patienten mit stark fixiertem Trommelfell oder bei Otosklerosepatienten wird man durch Blasen gegen das homo- oder das kontralaterale Ohr keine deutliche Impedanzänderung bekommen, wegen schlechter oder aufgehobener Mobilität in den schallleitenden Feilen. Diese Tatsache zeigt, dass der Luftstrom, der die Messbrücke trifft, allein keine falschen Ausschläge her vorruft, vorausgesetzt, dass die Tonsonde den Gehörgang gut schließt



Fig 5 Halbseltige Skizze eines horizontalen Schnittes durch den Nucleus tractus solitarius welche die terminale Ausdehnung der Fasern des n intermedius (facialis) n trigeminus n vagus und n glossopharyngicus im Nucleus darstellt. Die terminale Ausdehnung der verschiedenen Nerven ist durch verschiedene Symbole dargestellt (Nach A. TORNIK). Br p, brachium pontis; Cn, nucleus cuneatus; dm, nucleus dorsomedialis (Astruc); G VII, facialis-kern; caud, nucleus caudalis; n, n. fs, nucleus tractus solitarius; ip, nucleus intermedius; n, n. caud, nucleus caudalis; n, n. pr, nucleus principalis; n, n. motorischer Kern; n, n. VIII, motorischer Kern; n, n. VIII, Pyramidenbahn; rf, VIII, Afferente Fasern; n, VIII, sg, Substantia gelatinosa des Hinterhorns und nucleus caudalis; Tr sp, Tractus spinalis; n, n.

Nach einer Otoskleroseoperation ad modum Schuknecht kann man durch Blasen gegen das homolaterale Ohr relativ grosse rasche, phasische Impedanzänderungen registrieren. Diese müssen durch Kontraktion im m. tensor tympani verursacht sein. Der Grund dafür, dass die Ausschläge relativ gross sind, ist wahrscheinlich eine erhöhte Mobilität im Trommelfell und den restierenden Gehörknöchelchen. Bei Patienten, die ad modum Schuknecht operiert sind, kann man, wie erwähnt, durch Blasen gegen das kontralaterale Ohr selten Impedanzänderungen hervorrufen. Auch dies deutet darauf hin, dass der Tensorreflex in den meisten Fällen ungekreuzt ist.

Mögliche Reflexbahnen

Das Hautgebiet, von dem aus der „Blas reflex“ ausgelöst wird, ist von sensorischen Fasern folgender Nerven versorgt: n. facialis (n. intermedius), n. trigeminus, n. vagus und n. glossopharyngeus. An Versuchen mit Ratten hat Torvik (27) gezeigt, dass Fasern von diesem Hautbezirk im nucleus tractus solitarius enden (Fig. 5). Man glaubte früher, dass zu diesem Kern nur visceral afferente Fasern führten. Der Kern liegt in der reticulären Substanz und ist von einer Reihe Zellen mit kurzen Ausläufern umgeben, durch welche die Impulse wahrscheinlich auf die motorischen Facialis und Trigemuskkerne übergeleitet werden können (Brodal (2)). Bei Menschen sind die Verhältnisse wahrscheinlich im Grossen und Ganzen die gleichen wie bei Ratten.

SUMMARY

In man, the sensory stimuli from the external acoustic meatus, the pinna and immediate surrounding areas are transmitted centripetally through branches of the facial, trigeminal, vagus and glossopharyngeal nerves. In rats, the sensory nerve fibres from these areas terminate in the nucleus tractus solitarius. From this, the impulses may pass to the motor facial and trigeminal nuclei, probably by way of reticular substance cells.

By stimulating this area with an air jet, a clinically applicable method has been found of producing non-acoustical intra-aural muscular reflexes. The neural reflex path is probably similar to that in rats. Preliminary investigations indicate that the stapedius reflex is transmitted in both crossed and uncrossed paths, the tensor tympani reflex essentially uncrossed.

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Namdal Krankenhaus Namns

MASKING AUDIOMETRY WITH SELF RECORDING AUDIOMETER

I Normal hearing

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Results are reported in normally hearing listeners for the size of the threshold tracings in self recording audiometry in quiet, and in 45, 65, 83 and 103 db (re 0002 microbar) white noise. It is shown that the excursions diminish a few decibels in size when the recording is made in the presence of higher noise levels. As both the testing tone and the noise suffer the same per stimulatory loudness loss, the recorded tracings show no deterioration as a function of time.

In an earlier article Palva, Goodman & Hirsh (1959) reviewed the controversial literature on noise audiometry and presented evidence in favour of the validity of the critical band concept (Fletcher & Munson 1937, Hawkins & Stevens 1930) in normal and also in hard of hearing subjects. The masking stimulus employed was a white noise with an even spectrum from 20 cps to 7000 cps.

The measurements were made by presenting interrupted pure tone stimuli from 250 to 8000 cps to the listener first in quiet and then in the presence of varying levels of white noise. The thresholds were determined with the method of limits in all test conditions.

From the fact that differences in masked thresholds were exhibited neither by normal listeners nor by patients with various types of conduction and perception pathology—including two cases of verified acoustic tumors, Palva *et al.* concluded that masking audiometry in the form advocated does not make possible a differentiation of perception deafness into a cochlear and retrocochlear subgroup.

The introduction of self recording audiometry into routine clinical use has made other test conditions available. It is now possible to measure the size of excursions at threshold and—provided that the stimulation is made with a fixed frequency—the slope of the threshold tracing as a function of time.

With a self recording audiometer Bulger & Hirsh (1956) studied the masked thresholds in the presence of narrow bands (about 250 mels) of thermal noise. Five normal hearing persons served as subjects and all measurements were made with a continuously varying frequency. The tracings were recorded

over a frequency range of 150 to 6000 cps with interrupted test tones for masking over all levels 40, 60, 80 and 100 db. The amount of masking produced by the bands of noise coincided closely with the critical band widths reported by Harkins & Stevens.

Recently Jerger, Tillman & Peterson (1960) measured the masked thresholds in the presence of narrow bands of noise which were somewhat larger than those employed by Bilger & Hirsh. The method with adjustment with ordinary audiometers was employed using normal listeners as well as patients with conduction or perception deafness. Jerger *et al* subscribed to the conclusion of Palva *et al* that neither an impaired ear nor a normal ear can be differentiated on the basis of a masking audiogram in the presence of broad spectrum noise.

Bilger & Hirsh and Jerger *et al* found that there appeared masking outside the noise bands that is more masking than would be expected on the basis of the critical band widths. While Bilger & Hirsh reported this phenomenon in their normally hearing subjects, Jerger *et al* found it only in their perceptively deaf patients. Normal subjects and patients with conduction hearing loss showed masking curves predicted on the basis of the noise levels. This discrepancy is probably at least partly due to somewhat different characteristics of the testing system, e.g. the steeper rejection rate of filters used by Jerger *et al*.

In tests of masked thresholds in continuous noise the results reported earlier for per stimulatory fatigue must be taken into account. It has been shown by various investigators that a steady tone or noise heard continuously in an ear suffers a loss in loudness during the first few minutes of stimulation. This loudness loss can best be measured by presenting tones for loudness balance in the opposite ear; the physical difference in tones judged equal gives the amount of per stimulatory adaptation.

It may be asked whether the per stimulatory fatigue suffered by the ear due to continuous noise would affect the pure tone masked thresholds in this noise. This point was investigated by Egan (1955) who noted that although clear per stimulatory fatigue for the noise could be demonstrated by bilateral tests, no change in the masked thresholds as a function of the time appeared in unilateral masking tests. One might thus say that both the masked test tones and the masking noise suffered per stimulatory fatigue which did not affect the measured threshold values.

In this study we propose to measure two specific aspects of masking audiometry with the self recording technique. First we want to find out the effect of various masking levels upon the size of the threshold excursions. When the pure tone thresholds are measured in the presence of noise, the intensity area of the tones lies high above the thresholds in quiet. Thus the relative decibels corresponding to the size of the masked threshold excursions, a width of say 6-7 db, represent much larger intensities than the ear handles at similar excursions at threshold. The audibility of the tones might then rise faster and the results might show diminished excursions. This assumption

would be in line with the results of Riesz (1928) for the size of the intensity difference limen although there are no good reasons why audibility and size of the difference limen should necessarily be interrelated (Hirsh, Palva & Goodman 1954). Only as a limiting case can the self recording excursions be considered to have something to do with the intensity difference limen: the recordings representing the difference between the tones heard none of the time on the one hand and all of the time on other hand.

Secondly we want to follow the temporal pattern of the masked threshold using fixed frequencies during 3 minutes stimulation. Although Egan's results convincingly demonstrate that there is a parallel change in both the masking tone and the pure tone stimulus and thus that the recordings per se, are unaltered, our experience in adaptation measurements has often shown a large spread even in normal listeners. Thus it is necessary to have an accurate reference basis by the same technique as will be used later for hard of hearing listeners.

Experimental procedure

The Bekesy type self recording audiometer which was used as a basic unit provided an intensity regulation in 1 db steps over a range of 140 db in one minute. The test subject regulated the tone in the usual manner by pressing the key while hearing the tone and releasing it as soon as the tone disappeared.

The first part of the testing consisted in letting the patient first record his air conduction thresholds in quiet between 500 and 4000 cps. White noise was then turned on and the subject made threshold tracings over the same frequency range at three different noise levels. Each run took about 4 minutes and the total procedure for one ear thus lasted about 16 minutes.

The source of the white noise was the masking generator of the self recording audiometer which regularly operates through its own channel and conducts the tones to the non-tested ear. A mixing network was therefore provided to lead both the masking noise and the continuously changing pure tone into the same PDR 10 receiver. The noise had an almost even spectrum between 100 and 10000 cps and could be regulated in 5 db steps over all levels of 4, 6, and 8 db re 0.0002 microbar were used for masking.

Twenty three normally hearing ears were used in this experiment. The test persons included 3 highly trained subjects while the remaining 20 had very little if any training in auditory testing procedures.

The second part of the experiment was carried out using the three highly experienced subjects. A Peters SPD 2 audiometer was coupled into the tone channel of the self recording audiometer to provide a fixed frequency for continuous recording of threshold in white noise. Four different noise levels were used adding 10 db over all level to the three levels mentioned earlier.

The subject first recorded his threshold in quiet for the frequency being tested during one minutes stimulation. Thereafter the threshold recording

was made during a 3 minute test period with the noise on beginning at the weaker levels and proceeding systematically to the highest level. After the last test with 103 db noise masking was turned off and the subject recorded his post stimulatory threshold for a period of 2-3 minutes for evaluation of the remaining fatigue. The test thus lasted about 15 minutes at a time for each subject.

Similar recordings were made at the frequencies 500, 1000, 2000 and 4000 cps bilaterally but only one frequency was tested on any one day. The ear generally felt a bit dull after the testing but this sensation disappeared in about 15 minutes.

Results

Threshold excursions in noise for continuously changing tones

The size of the excursions was measured from peak to peak from the curves at several points for various frequencies and the mean size of the excursions was calculated from these figures. The values with the standard deviations are shown in Table I.

It is seen that the mean sizes of the threshold excursions in quiet are larger than the other means obtained from the excursions in noise; this is especially noticeable at the two higher noise intensities.

Calculation of the standard errors of the means showed that at 500, 1000 and 4000 cps the difference between quiet and 83 db noise mean excursions was 3 to 4 times the standard error. At 1500, 2000 and 3000 cps the difference was about twice the standard error.

The masked thresholds presented graphically in Fig. 1 conform fairly well to the critical bandwidths of Fletcher except for 500 cps where the curves reveal somewhat more masking than would be expected theoretically. Thus we ascribe more to the subject's searching of the true threshold at the beginning of the test run at 500 cps than to any real effect.

Threshold excursions in noise at fixed frequencies

While the frequency remains the same during the test period there are two variants in which changes can be observed, i.e. changes in the size of the excursions and fluctuations in the threshold level as a function of time.

TABLE I *Threshold excursions with continuously varying frequency*

Over all masking noise	500		1000		1500		2000		3000		4000	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Quiet	79	26	81	33	85	44	73	40	70	41	80	28
45 db	74	24	73	40	74	23	68	27	73	37	60	26
63 db	57	15	60	19	65	24	64	27	58	19	54	21
83 db	52	1	52	16	57	37	54	19	60	20	53	20

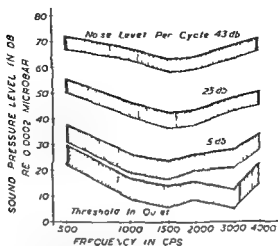


FIG. 1 The average size of excursions in quiet and in noise determined with a continuously changing frequency technique in a group of 23 ears. It is seen that the average size of the excursions is smaller in high level noise than in quiet. Apart from 500 cps the masked thresholds correspond well with the critical band widths.

Calculation of the mean value for the size of the masked thresholds was done in each of the six ears from four points in the masked curve: the initial size, and the sizes after 1, 2 and 3 minutes of listening in noise. The mean value of each frequency and their standard deviations are presented in Table II.

It can be seen at once that the size of the excursions in these trained ears is clearly smaller than in the group of mainly inexperienced listeners in table I. This is obvious especially at the higher end where the mean values deviate from each other by as much as 5 db. Also it can be noted that the experienced listeners are very consistent with their responses in all test conditions, which is apparent from the fairly small SD's.

Calculation of the standard errors of the means show that the masked mean threshold excursions deviate markedly from the mean excursions in

TABLE II: Threshold excursions with fixed frequency

Over all masking noise	500		1000		2000		3000	
	M	SD	M	SD	M	SD	M	SD
Quiet	56	15	59	11	49	23	29	09
45 db	45	08	47	09	34	09	25	06
65 db	41	08	39	09	31	07	23	05
85 db	38	07	35	07	31	05	21	05
105 db	40	09	34	13	28	06	22	04

ZUSAMMENFASSUNG

Bericht wird erstattet über bei Lauschern mit Normalgehör in der selbstregistrierenden Audiometrie erzielten Grossen der Schwelleaufzeichnungen bei Stille und bei weissem Geräusch von 45, 65, 83 und 103 db (re 0 0002 microbar). Es wird gezeigt, dass die Grosseabschweifungen einige Decibele weniger sind wenn die Aufzeichnung bei Vorhandensein höherer Geräuschniveaus bewirkt wird. Wenn beides der Versuchston und das Geräusch denselben reizmässigen Schallverlust erleiden, weisen die Registeraufzeichnungen keinen zeitfunktionsbedingten Schwund auf.

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DEAFNESS FOLLOWING MATERNAL RUBELLA

Retrospective and Prospective Studies

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A prospective study of children born to women who had had rubella in pregnancy during a widespread epidemic of the disease was instituted in 1951 and is still in progress. The present investigation comprises a prospective study of the hearing capacity of children in the Stockholm area who had histories of maternal rubella, as well as a retrospective study of children with hearing defects who had been referred to the Department of Otolaryngology and the Laboratory of Audiology, Karolinska Sjukhuset, Stockholm. The report also includes an inquiry conducted in the schools for the deaf and the special classes for hard-of-hearing children, with respect to pupils whose births were associated with the aforementioned epidemic.

INTRODUCTION

Since the report of Gregg (1941) on congenital cataract in infants born to mothers with rubella in pregnancy, numerous studies in this field have confirmed his findings. It is now a well established fact that congenital heart disease, developmental defects of the eyes, mental disorders, and impaired hearing are also among the abnormalities following maternal rubella. Hearing loss as a sequel of maternal rubella was first observed by Swan *et al* (1943). The literature on the subject has since assumed large proportions (Murray, 1949, Lancaster, 1954, and others). In Sweden the problem has been studied by Gronvall & Selander (1948) and Ivarstam (1951). Most of the investigations concerned with deafness have been retrospective—a fact which has led to somewhat controversial opinions on the risk of congenital abnormalities for children born to women with maternal rubella. However, in a recent prospective study by Jackson & Fisch (1958), who examined children 3–5 years old with histories of maternal rubella, it was found that out of 57 children whose mothers had had rubella during the first eighteen weeks of pregnancy, 14 suffered from hearing impairment. Two thirds of the cases were bilateral and nearly one-half were severe.

In Sweden relatively widespread epidemics of rubella occurred in 1944–45, 1950–51 and 1955–56. Data have been collected in regard to a number of

children born subsequent to maternal rubella the majority of them in the years mentioned above. The study reported here was prompted by the need for further information on the incidence and degree of deafness in children with histories of maternal rubella.

MATERIAL

A. In the years 1951 through 1959 a total of 752 children with impaired hearing, in most cases severe, were examined at Karolinska Sjukhuset. A history of maternal rubella was found in 92 of the children.

B. All schools for the deaf and special classes for hard of hearing children in Sweden were asked to report the number of their pupils born in 1951 or 1952. All the schools responded and reported 162 such children as of May, 1960, with notes on any known history of maternal rubella. The medical records as well as information on the children's ability to follow their lessons were obtained in the rubella cases.

Groups A and B constitute the retrospective part of the study.

C. The cases forming this group were selected from Lundstrom's nationwide study (1952) of the 1950-51 epidemic in Sweden. Forty six of 814 children with histories of maternal rubella in the first five months of pregnancy were known to live in the Stockholm area. Forty four of them were examined audiologically at the age of six to eight years. This group constitutes the prospective part of our study.

The three groups represent a total of 104 children with hearing loss due to maternal rubella. Some children are referable to more than one of the groups. Thus, 92 are represented in group A, 39 in group B and eight in group C.

METHODS

The audiologic examination was thorough, and special efforts were made to secure tone audiograms by means of play audiometry (Barr 1954). Reliable audiometric values were obtained in 91 of the 104 children. The hearing loss is expressed as the mean for the frequencies of 500, 1000 and 2000 c/s for the best ear, unless otherwise stated. Where exact determination by tone audiometry was impracticable—mainly in children born in 1956 or later—the hearing loss was estimated by informal hearing tests. Classification according to the degree of hearing loss was possible in all cases. The study is confined to perceptive hearing loss, in cases of combined defects only the perceptive component is reported.

Vestibular functions were examined by caloric and/or rotatory tests.

Although other examinations were performed, the results will be reported only insofar as they concern abnormalities of the eyes and the heart.

RESULTS

Retrospective Study

The children with severe hearing loss examined at Karolinska Sjukhuset in the period from 1951 through 1959 are grouped according to year of birth in Fig 1. The incidence of children with a history of maternal rubella varies between 0 and 40 per cent, indicating epidemics of rubella in 1944-45, 1950-51 and 1955-56. Histories of maternal rubella were found in 12 per cent, i.e. in 92 of a total of 752 children.

Among the 162 pupils in schools for the deaf or special classes for hard of hearing children 39 had histories of maternal rubella (25 per cent).

Degree of hearing loss

All of the 104 children from the three groups with definite perceptive hearing loss were classified according to the month of pregnancy in which rubella had occurred and the degree of hearing loss as shown in Table 1. Rubella was found to have occurred in the first to the fifth month of pregnancy. Only one case had been observed in the fifth month here, exact dates were given for the last menstruation period and the onset of rubella which was

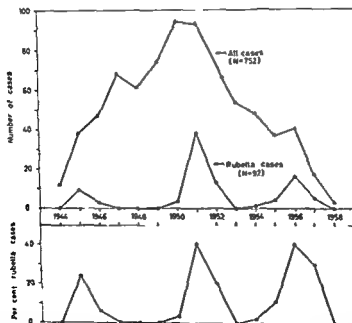


Fig. 1. Children examined at Karolinska Sjukhuset for severe hearing loss, grouped by year of birth. The incidence of rubella children ranges between 0 and 40 per cent, indicating rubella epidemics in 1944-45, 1950-51 and 1955-56. Of the total number of children 12 per cent had histories of maternal rubella.

TABLE 1. Degree of hearing loss in 104 children, grouped by month of pregnancy in which rubella occurred. The hearing loss is expressed as the mean for the frequencies of 500, 1000 and 2000 c/s in the best ear.

	Rubella in month of pregnancy no						Total
	I	II	III	IV	V	Unknown	
Number of cases	13	38	32	16	1	1	101
Hearing loss							
> 85 db	3	16	9	5		2	35 (31%)
55-84 db	6	17	17	8	1	1	50 (48%)
25-54 db	1	4	4	1		1	11 (11%)
< 25 db	3	1	1	3			7 (7%)
Unknown			1				1

the fifth day of the fifth month of pregnancy. In most cases maternal rubella had occurred in the second and third month. The highest degree of hearing loss was most common for rubella in the second month.

Birth weight

A large proportion of the children had been immature, 32 per cent having had a birth weight of 2500 g or less (Table 2).

Appearance of the tone audiograms

A typical audiogram of a hard of hearing child with a history of maternal rubella is a flat curve with small variations between the frequencies (Fig. 2). The degree of hearing loss usually differed considerably in the two ears, only a few cases showed nearly symmetrical audiograms. Two children had atypical audiograms, each of them showing high tone loss.

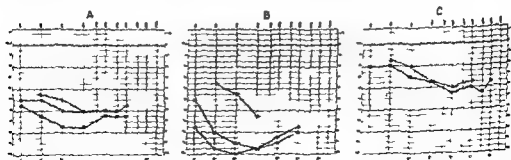


FIG. 2. Tone audiograms from three children with congenital rubella deafness. A, severe bilateral hearing loss. B, severe bilateral hearing loss (values for the bone conduction receiver are an expression of response to vibratory stimuli). C, total deafness right ear, moderate hearing loss left ear.

TABLE 2 *Ocular and cardiac defects and birth weights of the children with congenital rubella deafness*

	Rubella in month of pregnancy no						Total
	I	II	III	IV	V	Unknown	
Eyes							
Chorioretinitis	II	11	9	3			28 (30%)
Cataract		1	1				II (2%)
Not examined		2	3	5		1	11
Heart							
Pathologic	3	7	5				15 (16%)
Not examined		3	1	3		1	8
Birth weight							
< 2500 g	5	15	8	1		1	30 (32%)
> 2500 g	7	21	20	14	1	2	65 (68%)
Number of cases with hearing loss	13	39	32	16	1	4	104

Vestibular function

Ninety per cent of the children underwent rotatory and/or caloric tests. None could be registered as definitely abnormal.

Ocular defects

Cataract was observed in two cases. This condition leads to visual impairment and a further severe handicap for children with hearing defects. Thirty per cent of the children had chorioretinitis with no demonstrable visual impairment (Table 2).

Heart disease

Sixteen per cent of the children had congenital heart defects, nearly all of which had been treated surgically and most of which were well compensated (Table 2).

School rating

In addition to the 39 rubella cases in group B, school ratings were also obtained in 18 cases from group A. Despite the fact that the children were in schools suited to their hearing capacity, 18 of the 57 (32 per cent) were given poor ratings by their teachers.

Prospective Study

Forty-six children residing in the Stockholm area and born of mothers who had had rubella in one of the first five months of pregnancy were

TABLE 3 *Prospective study of 46 rubella children of whom 44 were examined at the age of 6 to 8 years. The hearing loss is expressed as the mean for the frequencies of 500, 1000 and 2000 c/s in the best ear*

	Rubella in month of pregnancy no						
	I	II	III	IV	V	I-IV	I-V
Number of cases	4	10	13	11	8	38	46
Normal hearing	2	7	11	9	7	26	36
Not examined				1	1	1	2
Hearing loss	2	3	2	1		8 (22%)	8 (18%)
Cases with severe hearing loss							
> 85-55 db		1	1	1		3 (8%)	3 (7%)

TABLE 4 *Rubella children with severe hearing loss attending schools for the deaf and special classes for hard of hearing children, and belonging to the nationwide prospective series. The hearing loss is expressed as the mean for the frequencies of 500, 1000 and 2000 c/s in the best ear*

	<i>Rubella in month of pregnancy no</i>							
	I	II	III	IV	V	VI-VII	I-IV	I-V
Number of cases	118	144	179	194	179	284	635	811
Cases with severe hearing loss								
> 85-55 db	2	12	7	5	1	0	20 (4%)	27 (3%)

subjected to this part of the investigation. Only two of the children missed the examination, their whereabouts being unknown. The results are set out in Table 3. A hearing defect has been defined as a loss of more than 20 db in two frequencies or more than 30 db in one frequency. This is the usual criterion of defective hearing in Swedish schoolchildren who undergo audiometric screening tests, and it also accords with the criteria used by Jackson & Fisch.

Of 38 children with histories of maternal rubella in the first four months of pregnancy, eight (22 per cent) showed hearing loss, and in three of these (8 per cent) the loss was severe. No case of deafness was found in children whose mothers had had rubella later than the fourth month of pregnancy. Three children showed a hearing loss of more than 55 db and one child a value between 25 and 55 db taking the best ear. One child had normal hearing in one ear and total deafness in the other. Two cases showed a hearing loss of no practical significance. One child had a tone audiogram with a slight

symmetrical loss in the high frequencies definitely differing from the audiograms of the other children. This child had been an asphyctic premature treated in an incubator for some time after birth.

DISCUSSION

Retrospective Study

The retrospective part of the study has shown as expected that rubella within the first four months of pregnancy may cause congenital perceptible hearing loss in the affected offspring. Such loss was found in one case to have resulted from maternal rubella as late as in the fifth month or the seventeenth week of pregnancy. A tendency to more severe hearing loss in cases with histories of maternal rubella in the second month is of interest, in view of the fact that most of the other abnormalities seem to result from maternal rubella acquired at the same stage of pregnancy.

The tone audiograms were similar in type to those described by other observers (Murray 1949; Fisch 1955; Jackson & Fisch 1960), i.e. characterized by a flat, sometimes gently sloping or rising curve. None of our cases showed total deafness. Atypical curves with pronounced high tone loss were observed in two cases. In one the loss might be attributable to anoxia, since the child had been a premature and asphyctic incubator baby. As prematurity is frequently a sequel of maternal rubella, the disease could be indirectly responsible for the hearing impairment in this child.

Severe damage of the vestibular apparatus was not observed in our cases. Although many rubella children are slow to stand and walk, this is probably due to other factors than abnormal vestibular functions.

Cataract, especially if bilateral, constitutes a further severe handicap in children with defective hearing. Only two of the 104 children had cataract, though one third of the series exhibited chorioretinal lesions. The existence of these lesions did not appear to affect the visual capacity.

Congenital heart defects concomitant with hearing loss as sequels of maternal rubella are frequently reported in the literature. In our series the incidence of congenital heart disease was 1.5 per cent. Surgical treatment had been successful in most cases and the heart disease was of little significance as an invalidating factor.

According to earlier reports, mental retardation not infrequently follows maternal rubella. The present series of children from schools for the deaf and special classes for the hard of hearing is a selected one limited to children regarded as fit for schooling. Severe mental handicaps are accordingly excluded. However, at eight to nine years of age, one third of the children were poor learners despite compensatory teaching for their hearing impairment. This accords with the findings of Hopkins & Kinzer (1949) who investigated 92 post-rubella deaf children and compared them with 61 deaf children whose mothers had not had rubella during pregnancy. The arrested

development of the rubella deafened children was attributed to general after effects of maternal rubella and not to the hearing loss *per se*.

In our series however, two thirds of the rubella deafened children attending special schools had satisfactory scholastic records. Of great importance is early recognition and treatment of the hearing loss the therapy consisting of hearing aids and special training methods which will usually preclude the development of a serious handicap.

Prospective Study

A prospective study of the incidence of hearing loss due to maternal rubella should preferably open with the recording of all known cases of rubella during pregnancy. The follow up should comprise the annotation of abortions as well as perinatal and later deaths and should conclude with audiologic examinations when the survivors have reached a suitable age. The same procedure should be followed for matched controls. In our study we first secured information on rubella during pregnancy from maternity hospitals. Surviving children with a history of maternal rubella within the first five months of pregnancy and known to reside in the Stockholm area were selected for study the selection presumably not affecting the validity of the results. Corresponding examinations of controls were omitted since facilities for the study were limited and in any case the incidence of perceptible hearing loss in a Swedish population chosen at random had been established by the investigation of Holmgren (1952). The latter found an overall incidence of perceptible hearing loss of 0.7 per cent among school children under conditions comparable with those in our prospective study.

Recently (1960) the British Ministry of Health published a report by Manson Logan & Loy on a nationwide controlled investigation of rubella and other virus infections during pregnancy. The incidence of deafness in children two years old who had histories of maternal rubella within the first four months of pregnancy was 2.7 per cent (five out of 183) compared with 0.09 per cent (four out of 3315) in the controls. The report by Jackson & Fisch (1958) concerns re-examination at three to five years of 57 children available from 79 survivors in the counties of London and Middlesex. They found hearing loss in 14 of the 46 children (30 per cent) who had histories of maternal rubella in the first four months. The corresponding figures for severe deafness were six of 46 (13 per cent). None had hearing loss from maternal rubella occurring later than the fourth month.

Under the auspices of the British Ministry of Health another group of the children was re-examined and reports made on 202 of 224 children with histories of maternal rubella within the first 18 weeks of pregnancy. Of these children 180 underwent re-examination at the age of three to seven years. Maternal rubella acquired within the first four months of pregnancy had led to deafness in 20 of 156 children (13 per cent) and to severe deafness in

eight of 156 (5 per cent). Here too, no cases with a history of maternal rubella acquired later than the fourth month of pregnancy were recorded.

The difference between the figures of Jackson & Fisch and those for children in other parts of the country may be due to the fact that in the former case the technique of examination was standardized but in the latter it was not. Jackson & Fisch examined only 27 of 79 children and did not state why the remaining 22 children were not available. Since it is likely that children with severe deafness are known and their parents more co-operative, deaf children were possibly over-represented in Jackson & Fisch's study and the aforementioned figure of 13 per cent for children with severe deafness may be too high.

In our study we had recorded 46 children from the Stockholm area who had histories of maternal rubella in the first five months of pregnancy. We were able to re-examine 44; two children not being traced. We therefore consider our results representative even though the series is relatively small. The incidence of hearing loss was 22 per cent for rubella within the first four months of pregnancy, with severe deafness in 8 per cent. For rubella of later onset no case of deafness was found.

Iundstrom in his study followed up children one to three years old comprising 1098 cases following maternal rubella. The incidence of deafness among the 635 children with histories of maternal rubella within the first four months of pregnancy was 2 per cent.

In the records from schools for the deaf and special classes for hard of hearing children a total of 27 cases with severe hearing loss (> 50 db for the best ear) were found to be referable to the above mentioned series (Table 4). This gives an incidence of 4 per cent, which is a minimum figure as some children may not be included.

At the present time, however, nearly all Swedish children are audiometrically screened during their first year at school. Thus, most cases of hearing loss are recognized and if the deafness is found to be severe, the children are referred to one of the schools included in our inquiry. It seems highly improbable that a child with a flat curve and a hearing loss of more than 50 db for the best ear (the criterion of severe hearing loss in this study) would be able to attend an ordinary school in Sweden.

Thus, the risk figure for severe hearing loss following maternal rubella within the first four months is 8 per cent, to judge by our findings in a small but thoroughly examined prospective series. The significance may be somewhat over-estimated due to the small number of observations, but the figure of 4 per cent in the larger series represents the minimum incidence.

A total of approximately 220 000 children were born in Sweden in 1911 and 1912 and of these 162 with severe deafness are attending the schools covered by our inquiry, indicating a risk of about 0.07 per cent for severe deafness. The estimated 4 to 8 per cent risk of severe hearing loss for children with histories of maternal rubella in early pregnancy is nearly a hundredfold greater. The total incidence of 22 per cent for hearing loss in the rubella

children may be compared with the 0.7 per cent for perceptive deafness found by Holmgren in Swedish schools

CONCLUSION

In a series of 752 children with severe hearing impairments the incidence of histories of maternal rubella was 12 per cent ranging between 40 per cent and zero according to birth in epidemic or nonepidemic years

All cases of congenital rubella deafness stemmed from maternal rubella in the first four months of pregnancy except one which was referable to the fifth month

The audiograms of rubella deafened children had a typical asymmetrical appearance, the curves being flat and the loss sometimes unilateral

Vestibular functions were usually normal

In schools for the deaf and special classes for hard of hearing children 20 per cent of the pupils born shortly after an epidemic of rubella had histories of maternal rubella in early pregnancy

The prospective study showed that the total incidence of hearing loss found among children born after maternal rubella occurring within the first four months of pregnancy was 22 per cent Four to eight per cent of the cases had severe deafness as compared with a figure of 0.07 per cent for children of corresponding age in the general population

ZUSAMMENFASSUNG

Im Jahre 1951 wurde eine prospektive Studie an solchen Kindern ins Werk gesetzt, bei welchen die entsprechende Gravidität der Mutter während einer ausgedehnten Epidemie in diese Zeit fiel Diese Studien befinden sich in fortgesetzter Bearbeitung

Die vorliegenden Untersuchungen befassen sich mit einer prospektiven Studie der Gehörkapazität von Kindern aus dem Stockholmer Gebiet deren Anamnese eine Rubeolaerkrankung der Mutter ergab Darüberhinaus umfasst sie eine retrospektive Studie an Kindern mit Gehördefekten welche an die otolaryngologische Abteilung und das Laboratorium für Audiologie des Karolinschen Krankenhauses überwiesen worden waren

Der Bericht umfasst des weiteren eine Umfrage durchgeführt an den Schulen für Taube und in den Spezialklassen für schwerhörige Kinder in besonderem Bezug auf die Schüler deren Geburtsdaten mit oben genannter Epidemie in Zusammenhang gebracht werden konnte

In einer Serie von 752 Kindern mit schweren Hörstörungen lag die Inzidenz mütterlicher Rubeolaerkrankung sub graviditate bei 12%, sie bewegte sich dabei zwischen 0 bis 40% im Zusammenhang mit der Geburt in epidemischen bzw nicht epidemischen Jahren

Alle Fälle kongenitaler Rubeolataubheit waren zurückführbar auf eine entsprechende mütterliche Erkrankung in den ersten vier Monaten der Gravidität mit einer Ausnahme für welche der fünfte Monat referiert wurde

Die Audiogramme rubeolatauber Kinder wiesen typisches asymmetrisches Vorkommen auf, mit flachen Kurven und gelegentlichem unilateralem Hörverlust

Die Vestibularfunktionen waren für gewöhnlich normal

In Schulen für Taube und Spezialklassen für schwerhörige Kinder konnten 25% der Schüler bezüglich ihrer Geburtsdaten in direkten Zusammenhang gesetzt werden mit mütterlicher Rubeolaerkrankung während der entsprechenden Gravidität

Die prospektive Studie zeigte, dass die totale Inzidenz für Gehörverlust bei Kindern, geboren nach mütterlicher Rubeolaerkrankung innerhalb der ersten vier Graviditätsmonate, bei 22% lag

Vier bis acht Prozent dieser Fälle wiesen schwere Taubheit auf, verglichen mit der Ziffer von 0.07% für Kinder entsprechenden Alters der gesamten Population

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TUBERCULOSIS OF THE MAXILLARY SINUS

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A case of tuberculosis of the maxillary sinus in a 49 year old woman who at the same time suffered from diabetes is described. The treatment consisted of a radical operation of the maxillary sinus and administration of streptomycin-dihydrostreptomycin PAS and INH which resulted in complete recovery. The importance of histological examination of inflammatory tissue and of bacteriological examination even in apparently unspecific common affections of the maxillary sinuses is emphasized.

Tuberculous processes in the accessory sinuses of the nose are very rare and not more than about 30 cases of tuberculosis of the maxillary sinus are reported in the literature. Twenty of these cases were described by Gleitsmann in a review as long ago as 1907.

In the majority of cases the tuberculous sinusitis seem to be secondary originating from a pulmonary or extrapulmonary source and spread either directly by means of the respiratory passages or through the lymph or blood vessels. In only 3 of the cases reported was there a tuberculous process in the maxillary sinus without evidence of tuberculosis elsewhere (Gleitsmann 1907).

Tuberculosis of the mucous membranes is in many cases thought to be caused by a tuberculous osteitis of the jaw bone which has disseminated into the mucosa of the maxillary sinus. This was held to explain 12 of the 20 cases reviewed by Gleitsmann (1907). According to the same author the mucosa of the maxillary sinus had in 5 cases been infected by a tuberculous affection in the alveolar process and in 2 cases a lupus in the mucous membrane of the nose had spread to the mucosa of the maxillary sinus.

The most important symptoms of tuberculosis of the maxillary sinus are (1) purulent secretion from the nose (2) nasal congestion (3) deteriorated sense of smell and (4) nasal bleedings. Pains, nausea, dizziness, lacrimation and visual disturbances may further occur in severe cases with osteitic changes in the orbit (Myerson 1956).

Pathologically 3 types can be distinguished (1) tuberculous lesions in the mucous membrane only (2) in addition to (1) specific osteitic lesions possibly including fistula formation and (3) tuberculoma. This type seems to

be extremely rare and has been reported only twice (Gleitsmann, 1907 Gibson & Prain 1954)

In its initial stages the disease may be difficult to diagnose unless tubercle bacilli are found in the nasal secretions or in the rinsing fluid from the maxillary sinus. In advanced cases with fistula formation and osteitic bone destruction diagnosis is easier, although in these cases the disease is often diagnosed as cancer. A biopsy should of course be made in all uncertain cases.

Earlier treatment consisted of radical and often mutilating surgical operations and the prognosis seems to have been most unfavourable. Writing in 1938 Hersch pointed out that of the 26 cases reviewed in the literature up to that year, only 6 had healed. The prognosis of tuberculosis in the accessory sinuses of the nose has however undoubtedly become much more favourable after the introduction of modern antituberculous therapy with streptomycin-dihydrostreptomycin, para amino salicylic acid (PAS) and iso nicotinic acid hydrazide (INH). The number of cases treated with these drugs is however, still very small. Single cases have been reported by Hara & Crane (1948) Gibson & Prain (1954) Oppenheim (1955) Ingberg (1957) and Kiviranta (1957). It therefore seems justifiable to report a single case of tuberculosis of the maxillary sinus which was treated surgically as well as with antituberculous drugs.

Case Report

The patient is the 49 year old wife of a labourer and she has upon the whole been in good health. In 1956 she suffered from three severe attacks of gall stones within a period of two months and a cholecystectomy was subsequently performed. A transient glucosuria was observed during the patient's stay in hospital.

The patient came to a consultation on Feb 6th 1959 complaining that for six months she had had a severe cold with abundant secretion from the left nostril which had been congested all the time. The examination revealed profuse purulent secretion in the nasal passages on the left side the middle turbinate being rather swollen and reddish. No polyps or ulcerations were seen. On the right side a slight reddishness of the nasal mucosa and some mucous secretion were observed. Posterior rhinoscopy showed purulent secretion in the left choana the right one being free. The epipharynx and the orifices of the Eustachian tubes looked normal as did the mesopharynx and larynx. Pus was obtained by puncture of the left maxillary sinus while the finding from the right sinus was negative. A combined procaine-penicillin-sulphonamide therapy was prescribed to the patient for a period of 10 days (500 000 units of procaine penicillin per day + sulphonamide Iederkyn® Iederle 0.5 per day). The left maxillary sinus was punctured every fourth or fifth day but neither subjective nor objective improvement was observed. A urine test showed 2% of sugar the glucose level of the blood being 295 mg%.

Because of these findings the patient was admitted to the Department of Internal Medicine of Vasa Central Hospital on April 20th 1959.

The diabetes responded well to metihexamide and the patient was discharged 9 days later adjusted to a maintenance dose of 0.05 g of metihexamide three times daily.

Punctures of the maxillary sinus were carried out during the hospitalisation and were continued for a few more weeks but the result was as poor as before. A radical operation of the maxillary sinus was therefore regarded as indicated and the patient was admitted to the Department of Oto Rhin. Laryngology of Vasa Central Hospital on May 21st 1959. The general state of the patient on admission was good. A roentgenogram of the thorax showed a heart of normal size and a slightly enlarged right latus but no pathological changes in the parenchyma of the lungs. Erythrocyte sedimentation rate 23 mm (1 hour). Haemoglobin 14.1 g%. Blood glucose 131 mg%. W. R. negative.

A roentgenogram of the accessory sinuses of the nose showed an intense shadow in the left maxillary sinus, the right sinus having a slight shadow due to marginal thickening of the mucous membrane. The ethmoidal cells and the frontal sinuses looked normal and filled. Anterior rhinoscopy showed nothing pathological on the right side but the middle turbinate on the left side was somewhat reddish and swollen. Purulent discharge was seen in the nasal passages. Posterior rhinoscopy showed purulent secretion in the left choana.

A radical operation (I use Caldwell's method) of the left maxillary sinus was performed on May 22nd 1959 in xylacaine anesthesia. The maxillary sinus was filled with pus and the mucous membrane was very swollen and fibrous. The latter was carefully removed and preserved for histological examination. No osteitic lesions in the sinus walls were noted.

The postoperative course was normal and the patient was discharged 5 days after the operation.

The histological examination of the mucous membrane removed from the maxillary sinus revealed a superficial epithelium consisting of layers of ciliated epithelium cells with spots of hyperplastic basal cells. The mucous membrane was thickened throughout as a result of the inflammatory changes. Most conspicuous were the granulomas formed by epithelioid cells and giant cells of the Langhans cell type. These granulomas occurred frequently in groups (Fig. 1). Here and there a central necrosis was seen in the granulomas. In sections stained by Eskelund's method a few acid fast rods resembling tubercle bacilli were seen. Examination in polarised light did not reveal any birefringent crystals or substances. The histological diagnosis was thus tuberculosis of the maxillary sinus.

After receiving the result of the histological examination antituberculous therapy was started with streptomycin dihydrostreptomycin 1 g every second day, PAS 12 g per day, and INH 0.3 g per day. The patient who already after the operation was relieved from her complaints came regularly for a

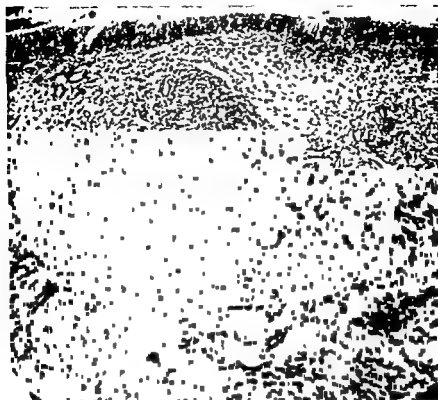


FIG. 1. Typical tuberculous changes in the mucous membrane of the maxillary sinus $\times 120$

check-up. Within 2 weeks of the operation rhinoscopy showed the nasal passages to be almost normal, and rinsing of the operated maxillary sinus resulted in only negligible amounts of mucus. The patient was given altogether 20 g of streptomycin-dihydrostreptomycin, after which the therapy was continued with PAS and INH. At the last check 13 months after the operation, the patient was completely symptom free. The rhinoscopy finding was normal, and the rinsing fluid from the operated maxillary sinus was quite clean. Erythrocyte sedimentation rate 3 mm (1 hour), blood glucose 185 mg%. The diabetes is under control, the patient receiving a daily dose of 0.5 g of Basilon[®] (Hoechst). A thorax roentgenogram still shows a somewhat enlarged right hilus, but no parenchymatic lesions in the lungs.

DISCUSSION

Although there are no certain signs of an open pulmonary tuberculosis, it seems very probable that the disease and the enlarged right lung hilus are related. It seems likely that this focus was activated by the development of the patient's diabetes, and that the verified tuberculosis of the maxillary sinus is of hematogenous origin. The clinical picture did not point either to

tuberculosis or a tumour, and the case would probably have been misdiagnosed had the removed mucous membrane of the maxillary sinus not been examined histologically. This case thus serves to stress the importance of an histological examination of all removed inflammatory tissue, even if the inflammation is apparently caused by an unspecific infection. Moreover, even in apparently unspecific infections of the nasal sinuses, a bacteriological examination of the secretion seems advisable. It is probable that tubercle bacilli would have been found in the rinsing fluid at the beginning of the treatment.

The favourable clinical course of this case seems to indicate that modern antituberculous treatment makes the prognosis of tuberculosis of the maxillary sinus much more promising than before

ZUSAMMENFASSUNG

Ein Fall tuberkulöser Kieferhöhlenentzündung bei einer 49-jährigen Frau, die gleichzeitig an Diabetes litt, wird rapportiert

Die Behandlung bestand in einer Radikaloperation der Kieferhöhle und antituberkulöser Behandlung mit streptomycin-dihydrostreptomycin, PAS und INH. Eine vollständige Heilung wurde erreicht. Die Bedeutung histologischer Untersuchung entzündeter Gewebe und die Wichtigkeit bakteriologischer Untersuchungen auch in scheinbar banaler, unspezifischer Nasenhöhlenentzündungen wird hervorgehoben.

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ETIOPATHOGENESIS OF SO CALLED IDIOPATHIC HEMOTYMPANUM AND ITS RELATION TO MORPHOGENESIS OF THE ADHESIVE PROCESS

*Chronic Postinflammatory Hematoma of the Pneumatic
Cavities of the Ear*

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■ summary will be found at the end of this article

A middle ear disease characterized by dark blue eardrum accumulation of coffee brown liquid in the pneumatic cavities of the ear and chronic conductive deafness is termed "idiopathic hemotympanum" in the medical literature. This condition is said to arise for unknown reasons as a consequence of nontraumatic bleeding into the middle ear cavities.

There is very little information about this disease in the literature. We have found 23 cases in all: 1 case from Sheppard (1914), 1 case from Vamola (1925), 2 cases from Shambaugh (1929), 1 from Fowler (1939), 2 from O'Donnell (1941), 1 from Simpson (1944), 2 from Ranger (1949), 2 from Menck Thygesen (1952), 2 from Johnson (1953), 1 from Simonon (1955), 3 from Sheehy *et al* (1956), 1 from Gignoux *et al* (1959), 2 from Streller (1959) and 1 from Navrátil (1960).

According to the literature the causes of the origin of the disease are quite obscure. Vamola believed that in his case bleeding into the tympanic cavity was caused by negative pressure following long term closure of the tube. Shambaugh and O'Donnell also thought that there was hemorrhage *ex vacuo*. On the other hand a second group of authors with Ranger at their head affirms that closure of the tube by itself cannot be the cause of idiopathic hemotympanum. Finally a third group, Johnson, Sheehy and Navrátil suppose that the disease arises from relapsing bleeding from varicose hemangiomas in the middle ear or from bleeding from some small spot in the mucous membrane of the pneumatic cavities of the ear which however has never been found at operation. All authors thus agree in the fact that the foundation of the disease is a hemorrhage but how this hemorrhage occurs remains still unexplained.

It is remarkable that in considering the etiopathogenesis of the disease the importance of inflammatory factors is completely omitted although an acute middle ear inflammation in the affected ear is mentioned in the history of most cases and in other cases where inflammation is not mentioned.

inflammatory anamnesis is suggested by findings of poorly developed pneumatization, destruction of the bone and sequestration in the region of the mastoid process found at operation, findings which would never be the results of simple bleeding.

This omission of the inflammatory factor can be explained by the fact that the clinical specialist does not usually have an opportunity to observe the whole development of the disease. He does not usually see the acute beginning but only the final condition which has developed after months and years and which is no longer reminiscent of an acute hemorrhagic middle ear inflammation. Thus a non-inflammatory idiopathic bleeding is assumed because no signs of inflammation are present at the time of examination while evident signs following bleeding are present: dark blue eardrum and coffee brown liquid behind it containing crystals of cholesterol and small clots of hemosiderin without cellular elements or only with their traces and shadows, the liquid being bacteriologically sterile.

The Authors' Own Observations

In 1951-1960 three patients with typical idiopathic hemotympanum were observed clinically. In none of them was blood disease present. We treated them conservatively, operated on them and finally examined the results of treatment. The observations made of them became the foundation of our new theory of etiopathogenesis of this still mysterious disease.

CASE 1—M O, boy, 6 years old. In November 1950 he fell ill with acute hemorrhagic otitis of the right ear. He was examined by a specialist who found hemorrhagic bullae on the eardrum, performed their scarification and administered detrition. Three days later the patient was without fever but new hemorrhagic bullae appeared on the eardrum and in the auditory canal for a period of 12 days. Then the eardrum was bluish and the condition did not change till February 1951. The patient was hospitalized and paracentesis was performed, after which some sanguinolent liquid flowed out. Penicillin was given. After this treatment hearing improved but the eardrum still remained bluish. A year later in November 1951 the patient was examined by the same specialist, dark blue eardrum and deafness were found. After catheterization dark brown secretion into the auditory canal occurred.

The patient was admitted to our department on the 30th of November 1951. Dark brown dry material could be seen in the right auditory canal, the eardrum was dark blue and bulged. He heard whisper at a distance of 2 m. The Schuller roentgenogram showed normal pneumatization bilaterally with diffuse clouding of the air cells but without destruction. Puncture of the ear drum yielded a yellowish brown viscous secretion and large amounts of coffee brown liquid were discharged from the little opening for a period of two days. Afterwards the eardrum was flattened, bluish gray but it began to bulge again. Anti-inflammatory roentgen irradiation of the tube and the middle ear was performed without success. Secretion from the

middle ear was sterile; contained albuminous coagulations and some disintegrating leucocytes

In January 1952 an atticotomy was performed on the right without plastic operation of the auditory canal. The external cortex of the mastoid process was thin in some places and translucent blue above some air cells. In all the pneumatic system in the antrum and in the tympanic cavity viscous coffee brown liquid was present; the mucous membrane was thin, pale and smooth and the intercellular septa were hard. The auditory ossicles were intact. Primary suture of the whole wound without drainage was performed. Secretion from the pneumatic cavities contained albuminous coagulations, shadows of erythrocytes and some leucocytes and lymphocytes. Microbes were found neither in smears nor by culture. Post operative healing was successful; the patient was released on the 15th day after operation with normal hearing. At a follow up examination eight years later in May 1960 the otoscopic finding was normal; hearing for whisper was 10 m, audiometric curve between +3 to -5 db.

According to clinical examination and operative finding the patient was suffering from coagulated hematoma which developed into acute hemorrhagic middle ear inflammation due to multiple episodes of bleeding from the mucous membranes of all the pneumatic cavities of temporal bone. Suppuration did not occur as the secondary infection was controlled by penicillin. Hemotympanum developed for 14 months; its manifestations were slightly noticeable immediately after subsiding of the hemorrhagic inflammation and became quite obvious twelve months later.

CASE 2—1 A woman 26 years old. The patient became hard of hearing in the right ear six years ago. She could not remember whether an acute inflammation preceded. Four months ago the deafness began to worsen and she experienced a feeling of pressure in the ear. She was examined by a specialist, then observed for a period of 4 months and at last she was sent to our department as Hemangioma cavi tympani de susp.

At her admission in April 1958 the right auditory canal was without pathological changes and dry. The eardrum was blue, flattened and without reflex. She heard whisper at a distance of 2 m. Puncture of the drum yielded coffee brown watery liquid. After this the hearing improved and blue colouring of the eardrum disappeared. For the following three months thin brown secretion continuously came out of the puncture opening. The eustachian tube was without pathological changes and large amounts of brown liquid were removed at every catheterization. In the roentgen picture after Schüller pneumatisation on the right was slightly arrested and clouded with signs of bone destruction. Cultures were negative.

The patient was released on the 15th day for family reasons; the hearing was good. Discharge of the brown liquid continued. Three months later she was admitted again because of repeated worsening of hearing and unceasing discharge. The eardrum was dark blue with bulging in the posterior upper quadrant. Mixed type deafness on the right was found audiometrically. In

July 1958 an atticotomy-mastoidectomy on the right with plastic operation of the auditory canal was performed. Below the thickened external cortex the air cells were filled to some extent with a thin coffee brown liquid containing crystals of cholesterol for the greater part however they were filled with solid brown tissue the colour of smoked meat as were the enlarged antrum and aditus. A rather large bone sequestrum was found in the connective tissue of the antrum and in the descending part of the sigmoid sinus. In the epitympanic cavity the mucous membrane was edematous brownish but the chain of ossicles was intact and easily movable. There was some brown liquid behind the thickened eardrum. Culture from it was negative. Histologically connective tissue was found with giant cell granulomatous reaction around the spaces left by crystals of cholesterol. In tissues granular pigment was found and identified histochemically as hemosiderin (Fig. 1). Postoperative healing was good and on the 21st day the patient was released. She heard whisper from 7-9 m and the audiometric curve showed reduced losses in the frequencies between 200 and 4000 from 30-75 db to 15-35 db.

At a follow up examination two years later in May 1960 the right eardrum was found to be intact thickened firmly attached to the promontory and immovable with Siegle's speculum. The eustachian tube was without pathological changes but after catheterization the hearing did not change. Adhesive process in the tympanic cavity was found with diminished hearing to the preoperative level.

According to the clinical examination and operative finding it was a hematoma of all pneumatic cavities of the ear which was partly coagulated and partly organized into connective tissue. The disease lasted 6 or more years. Finding of poorly developed pneumatization and the operative finding of bone sequestrum were suggestive of inflammatory origin of the disease. Histological findings pointed to hemorrhagic origin. The operation healed the hemotympanum anatomically but from the viewpoint of function it was not successful because the disease resulted in an adhesive process.

CASE 3—F. J. boy 19 years old. He fell ill in June 1959 with an acute middle ear inflammation on the right side. Spontaneous discharge from the ear ceased after two days. He was admitted to another Otolaryngologic Department where the eardrum on the right side was found to be hyperemically infiltrated and bulged in the posterior lower quadrant. He heard voice from 1 m. After paracentesis had been performed serosanguinous discharge with formation of fibrinous casts in the auditory canal lasted for several days. The patient was given penicillin from the fourth day chloramphenicol and later on tetracycline. Repeated culture was negative. As his condition was not improving he was sent to our clinic in July 1959.

On admission the eardrum on the right side was grayish pink with two protuberances in the posterior lower quadrant without discharge. Hearing for whisper at 6-8 m audiometric curve showed 15-45 db losses. This finding did not change for 3 weeks of hospitalization. In roentgenograms there was arrested clouded pneumatization on the right without signs of destruction.

The eustachian tube was without pathological changes. After discharge the patient was in permanent ambulant care. The eardrum findings and hearing worsened by degrees. The eardrum became grayish blue, blackish blue, bulging appeared in the posterior lower quadrant. Therefore the patient was admitted again at the end of the seventh month of the disease.

In January 1960 atticotomy with ossiculotomy on the right was performed with revision of the tympanic cavity but without plastic operation of the auditory canal. Large irregularly shaped air cells with hard intercellular septa were filled with solid connective tissue, the colour of smoked meat. Viscous brown secretion with crystals of cholesterol was found in the antrum and in the minute periantral cells. In the aditus and epitympanum there were grayish solid granulations which enclosed the epitympanic part of the chain of ossicles. Granulations also filled the region of the oval window. They were removed without interrupting the chain of the ossicles. The region of the round window and the lower part of the tympanic cavity were filled with brown secretion, after removing which two fine brown membranes were easily visible, extended between the long process of the incus and the promontory and between the manubrium of the malleus and the inner wall of the tympanic cavity. They were removed and thus the chain of ossicles was freed.

The liquid content was twice bacteriologically negative. The tissue filling the air cells was sent for histological examination. Finding reads as follows: connective tissue with numerous spaces, after dissolved crystals of cholesterol surrounded by giant cells. Grains of hemosiderin in the tissue (Fig. 2). Post-operative course was favourable, the bluish colouring of the eardrum disappeared by degrees, hearing improved to 10 m for whisper, the tube was without pathological changes, catheterizations were performed every other day. The patient was released on the 29th day after the operation. There was still a bluish tinge in the posterior lower quadrant of the eardrum, the audiometric curve in the frequencies between 500 and 4000 showed a 15 db loss.

At a follow-up examination 3 months later it was found that in spite of repeated catheterizations deterioration of hearing had occurred. The audiometric curve showed 10-20 db loss. The eardrum was bluish in the posterior half, the hemotympanum was not yet healed up and as there was concern that it would change into an adhesive process, the continuation of catheterizations was recommended.

The clinical examination and operative finding revealed hematoma in all the pneumatic cavities of the ear which for the most part was organized and to a lesser extent colliquated. The process began as a hemorrhagic inflammation which was intensively treated with antibiotics. Hemotympanum developed for 7 months. As early as after one month signs of hemotympanum were slightly visible and 6 months later the signs were typical. The eustachian tube was obstructed at the beginning, later on it was unobstructed.

Etiopathogenesis of the So called Idiopathic Hemolymphangium

On the basis of our observations and knowledge gathered from the literature the etiopathogenesis of the disease appears to us as follows:

1 *Acute hemorrhagic inflammation as the cause of bleeding into the pneumatic cavities of the middle ear and the mastoid process* From the era before the therapeutic use of antibiotics we have rich experience of how such an inflammation manifested itself: dark blue bullae appeared on the eardrum, there was serosanguinolent discharge from the ear, and in the most serious cases even pure blood was discharged. If trepanation was performed in the first 7–14 days, dark clots of blood were found in pneumatic cells of the mastoid process and the mucous membrane of the cells was hemorrhagic, while in the antrum signs of suppuration were already present, depending on the duration of the inflammation.

In our cases Nos. 1 and 3 the disease undoubtedly began as an acute hemorrhagic inflammation. In case No. 2 more detailed data are lacking, but the picture of involved pneumatization and the finding of two large bone sequestrs in the connective tissue point to an inflammatory origin. In the literature similar data suggest an inflammatory etiology as well, but no importance is attached to them in the origin of the disease.

Further development of such bleeding into the pneumatic cavities of the ear may continue in two ways. Formerly secondary suppuration occurred in most cases, which was treated by antromastoidectomy. In the era of antibiotics, however, the suppuration usually does not occur, and bleeding into the pneumatic cavities manifests itself by hemolymphangium after the acute influenza disease has subsided.

In the further interpretation of changes in the pneumatic cavities of the ear filled with clots of blood we relied on the work of Šikl (*Subdural Hematomas*, 1944) in which the author explains morphogenesis and the single stages of development of subdural bleeding. We believe that these hematomas resemble in many points—except for the cause of their origin—our idiopathic hemolymphangium.

2 *Changes in the pneumatic cavities of the ear filled with blood* Blood effused into the open cavities of the ear in an acute hemorrhagic inflammation coagulates in about 24 hours and if under the influence of antibiotics or for some other reason secondary suppurative infection does not occur, regular changes occur in blood coagulations in two forms, of which the first is *organization of the coagulum*. Between the 7th and the 21st day solid red brown casts form in the air cells and fibroblasts and capillaries from the injured cells begin to grow into them. This process was distinctly marked in our cases Nos. 2 and 3 in which formation of connective tissue occurred pervaded with products of disintegration of erythrocytes, i.e. with numerous foci of hemosiderin and crystals of cholesterol. During the organization of the surface layer of the coagulations another process occurs more deeply, namely *autolysis of the fibrinous part of the coagulum*, which leads to the stopping of

growth of fibroblasts and to formation of connective tissue. In cases Nos. 2 and 3 we observed also partial coagulation of the coagulation along with organization. In case No. 1 we saw the process of coagulation to the whole extent as the processes of organization did not occur at all perhaps for the reason that there was no serious damage to the epithelium.

3. *Influence of patency of the eustachian tube* in the origin and development of the disease is undoubtedly problematic. During the acute episode ventilation of the middle ear by the tube is undoubtedly impossible. Later on when hemotympanum has already developed, patency of the tube has no great influence on the development and duration. Many authors take a sceptical attitude towards the question of patency of the tube. They allege that hemotympanum arose while the tube was patent and persisted in spite of catheterizations performed later. In our cases too the tube was unobstructed but the question arises as to whether there was valve closure in the tympanic orifice, caused by swelling of the mucous membrane and by pressure of the accumulated viscous secretion. In all our cases the eardrum was bulged in the posterior half under the influence of pressure of the secretion which is never seen in simple transudations *ex vacuo* from the obstruction of the tube in subacute secretory catarrhs. But in developed hemotympanum the patency of the tube has a secondary importance in the course of the disease as well as a parenthesis because as we shall show later outflow of liquid products of chronic hematoma alone will not lead to its healing.

4. *Overpressure in the tympanic cavity* in idiopathic hemotympanum caused by the liquified hematoma is explained in a similar way to the enlargement of a subdural hematoma in its later development when after a period of latency the hematoma grows to such an extent that it begins to oppress the brain and to manifest itself by both general and topical cerebral signs. Siki mentions three possible causes of growth of a chronic subdural hematoma:

(a) Bleeding from dilated capillaries of the granulation tissue which is an expression of venous congestion.

(b) Diffusion of liquid into the sac of the hematoma which was proved experimentally by Gardner in 1932. Diffusion is conditioned by the difference in the albumin content in the liquid of the hematoma and the surrounding tissues causing a great difference in osmotic tension between both these media by the action of which the liquid diffuses from the liquor and tissues into the sac of the hematoma.

(c) Inflammatory exudation into the sac toxic products arising in autolysis of a chronic hematoma irritate the granulation tissue to develop an inflammatory reaction which causes an increase in permeability of the capillary walls for all components of the fluid.

Also the rise and growth of post-traumatic bone cysts the foundation of which is also a chronic hematoma is explained by similar processes.

There is no doubt that the same processes occur also in a postinflammatory chronic hematoma of the cavities of the temporal bone. Osmotic absorption

of liquids from the neighbouring tissues, bleeding from venous lacunae of granulation tissue and toxic injuries of capillaries cause overpressure in the ear cavities, bulging of the eardrum, its anesthesia, atrophy and in rare cases also spontaneous perforation. If we perform a puncture of the tympanic membrane and remove the coffee coloured contents, protrusion of the eardrum disappears immediately and hearing improves to normal. After puncture, however, the opening usually closed and soon a new bulging occurs, as in our cases Nos. 1 and II.

Our case No. 2 after one puncture brown liquid secreted continuously for three months and the discharge was stopped only by operation. But this was a very old hematoma lasting 6 years or more, with watery content diluted by absorption of liquids from the neighbouring tissues and flowing out perhaps even through an unobstructed eustachian tube. In spite of permanent discharge the eardrum bulged in consequence of overpressure.

It can be seen from our observations that the liquid in the hemotympanum after removal and after paracentesis was always formed and supplied again and again by the same processes as in subdural hematoma. It is very unlikely that the new formation of the liquid of autohysed coagulum can be explained by relapsing bleeding from one vessel or varix, as Johnson, Sheehy and Navrátil tried to do.

Some authors saw retracted eardrums in their cases. But this does not argue against overpressure accumulation of liquid in the pneumatic cavities of the ear. Sheehy's case 3 had a retracted eardrum, but in paracentesis its content flowed out under pressure, as the author expressly mentioned. We believe a retracted eardrum in idiopathic hemotympanum to be a sign of presence of adhesive scars in the tympanic cavity, which prevents the drum from bulging.

a. Healing of the so called idiopathic hemotympanum. Chronic hemotympanum can be healed by punctures, paracenteses and catheterizations only rarely in those cases where the disease is limited to the tympanic cavity and the whole coagulum colligated there. Thus it might have been in Vymola's case. In the other cases, that is, in the majority, conservative treatment cannot be successful. Hemorrhagic inflammations affect most frequently ears with normal pneumatization and bleeding occurs not only into the tympanic cavity, but also into all the pneumatic cavities of the ear. When regular changes in the hematoma occur, i.e. its organization, on the one hand, and colligation on the other, it is conceivable that neither puncture nor paracentesis will do for treatment of the affected cavities. In all our cases we tried this treatment without success and other authors met with the same failure.

The definitive treatment and its result depend undoubtedly on the extent to which colligation on the one hand, and organization of hematoma, on the other, occurred. If full colligation of the fibrinous part of the coagulum occurred, as it did in our case No. 1, the conditions in the middle ear may return to normal after atticotomy, as we found in our case after 8 years.



Fig. 1. Connective tissue with numerous spaces after dissolved crystals of cholesterol surrounded by giant cells. Grains of hemosiderin in the tissue.

But if besides autolysis also organization of the coagulum occurred as in our cases Nos 2 and 3 the situation is much worse. At operation on the third case we came to the conclusion that we were operating on an early stage of an adhesive process: in the air cells of the mastoid process we found tissue the colour of smoked meat while in the epitympanum there were grayish rosy masses of connective tissue which differed little from the connective tissue in an adhesive process. In addition to it we found in this case two membranes partitioning the tympanic cavity. These forms of hemotympanum in which connective organization of hematoma prevails result at last in a real adhesive process. Anatomic healing of the hemotympanum occurred in our case No 2 as we found in the follow up examination after two years. In case No 3 the hemotympanum has not healed in the fifth month after operation and there is concern that it will change into an adhesive process in the tympanic cavity. Several patients of other authors also developed adhesive processes in the end.

Supplementary Material on Inflammatory Etiology of Idiopathic Hemotympanum

At the conclusion we shall mention some phenomena which we meet with in the pathology of the ear all or some of the elements of which remind us of the picture of idiopathic hemotympanum. Some findings in chronic middle ear inflammations and conditions after inflammations belong to this group. We mention them in order to point to general laws and the relations of various diseases and to lend support to the inflammatory etiology of hemotympanum.

1 *Postinflammatory hemoepitympanum*. F. M. woman 18 years old. From early childhood she suffered from bilateral chronic middle ear inflammation with deafness. In the left ear the whole eardrum was absent and only a shortened manubrium of the malleus projected into the open space of the tympanic cavity. A black blue downwards protruded sac could be seen behind it. In trepanation coffee brown liquid and crystals of cholesterol were found in the atticocanal cavity. A tuft of tough connective tissue the colour of smoked meat was found in the aditus. The tympanic cavity was separated from the epitympanum by a membrane which formed the blue sac visible at otoscopy. We diagnosed the disease as hemoepitympanum as all components of idiopathic hemotympanum were present: coffee brown sterile liquid with crystals of cholesterol and grains of hemosiderin in histiocytes and typical connective tissue with spaces after cholesterol and small clots of hemosiderin (Fig. 3). Inflammatory etiology was here without doubt. There was only the difference that the whole eardrum was absent.

2 *Residues of idiopathic hemotympanum in matured adhesive processes*. In 4 out of 42 adhesive processes operated on we found quite distinct residues after hemotympanum both macroscopic—coffee brown liquid and connective tissue the colour of smoked meat—and microscopic—connective tissue with

spaces after crystals of cholesterol with giant cell reaction and foci of hemosiderin (Fig. 4). The adhesive process developed here owing to organization of postinflammatory hematoma in the tympanic cavity.

Thus, on the one hand, we saw direct change of idiopathic hemotympanum into an adhesive process (our case No. 2) and on the other we could see the residues of hemotympanum with which the process once began in some mature adhesive processes. We consider this disease to be one of the causes of morphogenesis of an adhesive process.

3. Cystic change of the air cells of the mastoid process in chronic middle ear inflammation with cholesteatoma. In 13.3 per cent of chronic middle ear inflammations operated on we found residues of chronic hematoma with cholesteatoma, coffee brown liquid with crystals of cholesterol and in some cases also connective tissue the colour of smoked meat.

Here is obvious the continuous series of developmental signs of idiopathic hemotympanum changing into an adhesive process on the basis of which cholesteatoma itself may develop under certain conditions and at a still later stage. The fact that some cholesteatomas develop on the foundation of an adhesive process was already known to Witmarck (1918). In some cases observed by us all three processes were present at the same time: residues after idiopathic hemotympanum, adhesive changes and cholesteatoma, which developed as the latest change on the basis of an adhesive process preceded by idiopathic hemotympanum.

At the conclusion of our work we want to state our opinion about the nomenclature used up to now, which we do not consider to be correct. The term used by American authors, *blue eardrum*, is quite unacceptable, as it does not give a true picture of the essentials of the disease. The other term, *idiopathic hemotympanum*, which is more frequently used and which was accepted by Sheehy after a discussion in which he stated that we know nothing about the etiology of the disease, expresses the state of our knowledge up to now. But this state is changing essentially through new information, which we have summarized in this work, and therefore we propose a new term: *chronic post-inflammatory hematoma of the pneumatic cavities of the ear*. This term includes the inflammatory etiology of the bleed in, the chronic course and extent of the anatomic region into which the bleeding occurs.

SUMMARY

The authors present a new theory about the origin, development and consequences of idiopathic hemotympanum, which they have worked out on the base of the analysis of three cases of their own, study of 23 cases from literature and various supplementary materials.

The origin of the disease is an acute hemorrhagic inflammation of all pneumatic cavities of the ear. If secondary suppurative infection of blood coagulations does not occur because of treatment with antibiotics, regular processes occur in the coagulum: (1) organizing processes producing typical histological findings; (2) autolytic processes

producing a coffee brown liquid by coagulation of the fibrinous component of the coagulum, again with typical microscopic findings

Overpressure of liquids in the pneumatic cavities manifests itself by bulging of the eardrum. The overpressure is conditioned by the absorption of liquids from the neighbouring tissues in consequence of the great colloido osmotic difference of two inner media: the hemotympanum liquid and the liquids of the neighbouring tissues. Also transudation from venostasis and inflammatory exudation from granular tissue may take part in these very active biophysical processes.

Patency of the eustachian tube has no influence on the development and duration of hemotympanum. Puncture of the tympanic cavity and paracentesis have no effect either, because the liquid removed is regenerated and newly supplied by the biophysical processes described.

Healing requires atticotomy with revision of all pneumatic cavities of the ear. Cases in which the whole hematoma is coagulated can be healed. Cases in which the organizing factor is expressed tend to result in an adhesive process.

The authors propose a new term instead of the term used up to now, viz. chronic postinflammatory hematoma of pneumatic cavities of the ear, which corresponds to etiopathogenesis and localization of the disease.

ZUSAMMENFASSUNG

Autoren legen eine neue Theorie über die Entstehung, Entwicklung und Folgen des idiopathischen Haemotympanons vor. Dieselbe ist auf Grund einer Analyse von 3 eigenen Fällen, des Studiums von 23 Fällen aus der Literatur und verschiedenen Ergänzungsmaterials bearbeitet worden.

Die Ursache der Krankheitsentstehung ist eine akute hämorrhagische Entzündung sämtlicher Lufträume des Ohres. Kommt es unter Einfluss der antibiotischen Behandlung nicht zu einer sekundären eitrigen Infektion des Blutkreislaufs, verlaufen im Koagulum gesetzliche Vorgänge: a) Organisationsvorgänge führen zur Bildung eines schleimfleischfarbigen Bindegewebes mit einem typischen histologischen Befund; b) autolytische Vorgänge bilden durch Kolligation des Fibrinbestandteiles des Koagulums eine kaffeebraune Flüssigkeit wiederum mit einem typischen mikroskopischen Befund.

Der Überdruck der Flüssigkeit in pneumatischen Räumen ist aus der Vorwölbung des blauen Trommelfells ersichtlich; derselbe ist durch das Ansaugen der Flüssigkeiten aus den umliegenden Geweben infolge des grossen kolloidal osmotischen Unterschieds von zwei inneren Milieus bedingt und zwar der Flüssigkeit des Haemotympanons und derer Nachbarorgane. An diesen sehr aktiven biophysikalischen Vorgängen kann auch die venostatische Transsudation und die entzündliche Exsudation aus dem Granulationsgewebe teilnehmen.

Die Durchgängigkeit der Ohrtrompete ist für die Entwicklung und Dauer des Haemotympanons ohne Bedeutung; auch die Punction der Paukenhöhle und die Parazentese ist zwecklos, da die abgeflossene Flüssigkeit durch die angeführten biophysikalischen Vorgänge fortwährend erneuert und ergänzt wird.

Die Heilung erfordert die Attikoantromastoidektomie mit der Revision sämtlicher Lufträume des Ohres. Die Fälle, wo das ganze Hämatom flüssig geworden ist, können bis zur Norm heilen. Fälle mit ausgeprägtem Organisationsbestandteil haben eine grosse Neigung in einen adhäsiven Prozess auszumünden.

Verfasser legen anstatt der bisherigen Benennung eine neue vor, und zwar

chronisches nachentzündliches Haematom der pneumatischen Räume des Ohres, die der Etiopathogenesis und der Lokalisierung der Erkrankung entspricht.

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AIR FLOW PATTERNS AND CILIARY ACTIVITY IN THE TRACHEA AFTER TRACHEOTOMY

A Method of Determination in vitro of the Rate of Ciliary Beat in a Tracheal Model

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An experimental method for studying the rate of ciliary beat on pieces of respiratory mucous membranes exposed to artificial to and fro ventilation has been evolved and tested.

The influence on the rate of ciliary beat exerted by some aerodynamic factors just below a constriction (e.g. the larynx or a tracheal cannula or a pathological stenosis) in the airways is demonstrated.

The efficiency of heat and moisture exchangers (HME's) used in post-tracheotomy care appears from their influence upon the function of respiratory mucous membranes.

It is also shown that ciliary activity, which has been stopped by drying for a short time, can be regained with the use of a HME.

Finally, the present method seems well suited for testing the influence exerted by single physical factors and also by chemical substances on the rate of the ciliary beat of respiratory mucous membranes.

The rate of mucous flow and ciliary activity has been widely used in different biological experiments. As early as 1933 the rate of ciliary beat in excised pieces of respiratory mucous membranes was studied and recorded with a microscope and a motion picture camera by Froetz and later on by several other authors. A similar device with a working distance allowing clinical *in vivo* studies was described by Frenchner & Richter in 1939. The first experimental method for objective recording and determination of the rate of ciliary beat *in vivo* as well as *in vitro* was devised by Dahlmann in 1955, 1956 and 1960. This method adequately reflects physiological conditions within the trachea of animals during normal nasal and oral respiration.

In vitro determinations of the ciliary function, however, have always been regarded as unreliable, partly because the mucous membrane must be detached from its innervation and blood supply and partly because chemical and physical influence exerted by the respired air on the mucosa is eliminated. The latter disadvantage can easily be compensated for experimentally but hitherto such a method has not been worked out.

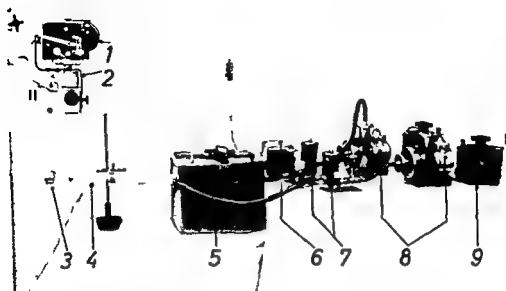


FIG. 1. The experimental equipment. 1 camera, 2 microscope, 3 heat and moisture exchanger, 4 tracheal model, 5 saturator, 6 magnetic valve-changer, 7 electro-magnetic valves, 8 blowing and sucking fans, 9 variable auto-transformer.

An equipment for comparative *in vitro* studies on pieces of excised respiratory mucous membranes exposed to experimental to and fro ventilation will be described in this paper. The method involves continuous control of (a) the ventilatory frequency, (b) the tidal volume, (c) the temperature and (d) relative humidity of the respired air. Furthermore the degree of contact between the respired air and the inner wall of a tracheal model in which the specimens were placed can be evaluated (Ingelstedt och Toremalm, 1960a).

As a consequence of the results obtained the function of the tracheal mucous membranes in man after tracheotomy will be discussed with special regard to pathological air flow patterns in the trachea and the use of previously described heat and moisture exchangers (HME's) for prophylactic treatment during the postoperative period (Toremalm 1960a, b).

EXPERIMENTAL EQUIPMENT

Principle

The ciliary activity was experimentally studied on specimens from respiratory mucous membranes which were exposed intermittently to fully saturated air at 37°C in one direction and to cooler and drier air (ordinary room air and room air after passing a HME) in the other direction in a

TABLE I *The rate of ciliary beat of mucous membrane pieces related to the time they have been exposed to artificial ventilation in a tracheal model provided with an inlet constriction closely related to a tracheal cannula*

Figures in italics are means

Rabbit no	Time after death, min	At start	Beats per minute						
			Time of exposure to artificial ventilation minutes						
			5	10	12	15	18	20	25
2	30	413	291		167			166	139
		413	300		163			157	139
		413	295	—	163	—	—	157	125
		413	300		163			166	139
		<i>413</i>	<i>297</i>		<i>164</i>			<i>162</i>	<i>136</i>
2	45	536	254		133				
		509	260		133				
		533	266	—	133	—	—	—	—
		536	266		139				
		<i>523</i>	<i>262</i>		<i>134</i>				
2	115			210		157			
				231		155			
		—	—	255	—	153	—	—	—
				231		153			
				<i>210</i>		<i>155</i>			
2	135	452		284		252			
		455		284		252			
		462	—	288	—	252	—	—	—
		435		284		252			
		<i>451</i>		<i>285</i>		<i>252</i>			
3	30			273		210		151	
				273		267		151	
		—	—	273	—	215	—	151	—
				273		240		151	
				<i>273</i>		<i>213</i>		<i>151</i>	
4	45	300		190		125	100		
		300		190		125	99		
		300	—	178	—	123	100		—
		300		178		123	100		
		<i>300</i>		<i>181</i>		<i>121</i>	<i>98</i>		
5	30	171	76						
		166	80						
		167	76	—	—	—	—	—	—
		160	66						
		<i>166</i>	<i>75</i>						

TABLE I (cont)

Rabbit no	Time after death min	At start	Beats per minute						
			Time of exposure to artificial ventilation, minutes						
			5	10	12	15	18	20	25
3	40	380	218						
		375	218						
		375	223	—	—	—	—	—	—
		387	233						
		379	203						
3	100	500	231						
		470	231						
		479	229	—	—	—	—	—	—
		500	223						
		490	230						
5	19.5	639	166						
		629	167						
		629	167	—	—	—	—	—	—
		663	167						
		640	166						
5	270	610	333						
		615	333						
		645	333	—	—	—	—	—	—
		632	325						
		611	331						
5	385	500	353						
		500	353						
		500	363	—	—	—	—	—	—
		500	363						
		500*	358						

* Three minutes after start

Tracheal model The rate of the ciliary beat was determined indirectly by recording synchronously appearing surface reflexes on the mucosa, which were caused by the vertical light beam from a microscope

Model

The outgoing air from an experimental respirator device (Fig. 1) was warmed and moistened in a saturator to 17°C and a relative humidity of 100%, and then blown (expiratory phase) through a tracheal model, made of plexiglass, intermittently 16 times per minute at a 'tidal volume' of 600 ml by a fan which could be regulated. Interchangeably equal volumes of room air (20–21°C, relative humidity 50–55%) were sucked (inspiratory phase) in the other direction. The model was double-walled for the purpose of in-

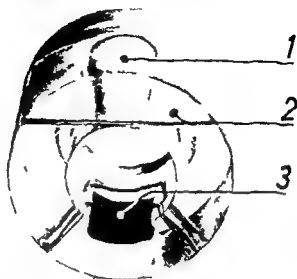


FIG. 2. The plexiglass inset with the channel for filming (1) which is separated from the inner lumen by a thin cover glass (2) and at the bottom (3) the groove in which the specimens were placed.

sulation and its inner diameter was 16 mm. The temperatures of the room air were determined with a psychrometer before and after each experiment. At the free end of the tracheal model there was a ring shaped plexiglass inset (Fig. 2) with the same inner diameter. At the bottom of this inset there was a groove in which the specimens were placed on a level with the inner surface. The model was then provided with a smooth circular constriction (Fig. 3) with an inner diameter of the same size as that of an ordinary tracheal cannula (10 mm in diameter). Finally a HMI with a moisture recovery efficiency of about 50% could be adapted to this constriction as seen in Fig. 4. In this case the temperature and relative humidity of the inspired air could be estimated at about 30°C and the relative humidity at about 80% in accordance with an earlier investigation (Torfvalm 1960a). The mucosal specimens were filmed at intervals (see Table I and II) through a channel (1, 2 above) which was separated from the lumen of the model by a thin cover glass in order to prevent the respired air from escaping and entering this way.

Microscope

An ordinary Zeiss operation microscope (Otoscope) provided with a separate tube for moving picture recordings with a magnification of up to $\times 80$ was used. The working distance was 20 cm and no connections between the microscope and the model were needed.

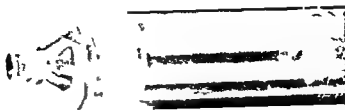


FIG. 3. Tracheal model provided with an inlet constriction.

Camera

The moving light reflexes on the mucosa were recorded on a 16 mm negative film (Gevaert 30) by a Bolex H16 R₁₁ VS moving picture camera at a rate of 64 frames per second.

COMPUTATION OF THE RATE OF CILIARY BEAT

Later on the film strips were projected with the aid of a Zeiss projector and the rate of the ciliary beat was calculated by two trained laboratory workers independently of each other in accordance with the method devised by Dalhamn in 1955 as follows.

The number of frames released by the projector in a second and the time taken to count 50 ciliary beats when the film was projected was measured. If this time was t seconds and the rotation speed of the projector 15 frames per second the rate of ciliary beat x per minute could be computed in the following manner:

$$x \text{ beats/min} = \frac{50 \times 60}{t} \text{ beats/min} \\ (15 \times 60 \text{ exposures/min}) = \frac{15 \times 60 \text{ frames/min}}{1}$$

MATERIAL

Rabbits with a body weight of about 2 kg were killed without anaesthesia and thereafter their tracheas were removed. Care was taken to keep the tissues moist in a glass tube placed in a thermos flask containing warm water at a temperature of 37°C. Specimens were then cut from the tracheas and in every specimen the membranous part was removed so that all determinations were made on the anterior tracheal wall. About 30 minutes after death



FIG. 4. Tracheal model provided with an inlet constriction and inlet filtration with a heat and moisture exchanger.

the first specimens of each trachea were exposed to artificial ventilation. The present experiments were planned in three series in order to attempt an answer to the following clinical questions:

(1) How does respiration through an ordinary tracheal cannula affect the ciliary activity in the lower part of the trachea after tracheotomy during normal room air conditions?

(2) What is the effect of using a heat and moisture exchanger (HME) during the same conditions?

(3) Is it possible to regain lost ciliary activity with the aid of a heat and moisture exchanger (HME)?

RESULTS

I. Tracheal model with constriction

The single determinations and mean values of the rate of ciliary beat at varying lengths of time (5 to 25 min) during which the specimens were exposed to the artificial ventilation appear from Table I. The specimens were taken out from the thermos flask 30 to 585 min after the rabbits were killed. The mean values are plotted on a diagram (Fig. 5) for comparison with the series in which a HME was used.

II. Tracheal model with constriction and a HME

The single determinations and mean values of the rate of ciliary beat at varying lengths of time (5 to 150 min) during which the specimens were exposed to the artificial ventilation, appear in Table II. (In several cases the

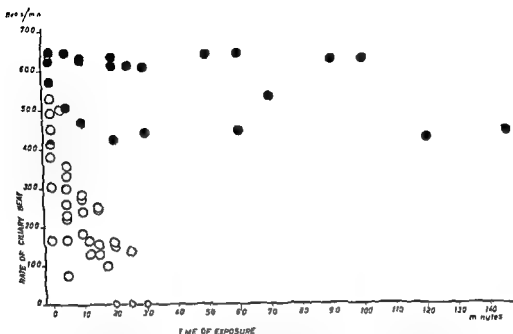


FIG 5 The rate of ciliary beat in beats per minute related to the time during which the specimens were exposed to artificial ventilation. Rings = a constriction (10 mm in diameter) placed on the inlet of the tracheal model. Points = the constriction was provided with a heat and moisture exchanger.

rate of the ciliary beat was over 500 beats per minute also after an exposure time of 480 min.) The specimens were taken out from the thermos flask 30 to 240 min after the rabbits were killed. The mean values are plotted on a diagram (Fig. 5).

III Tracheal model with constriction and on and off use of a HME

In these experimental series the rate of ciliary beat was recorded in the same manner as that mentioned above. In addition, direct observations in the microscope were carried out every minute for 20 min. after removal of the HME, as it was not possible to determine exactly the moment when the ciliary activity ceased. From the beginning a HME was used (ON) but at intervals it was disconnected (OFF). The results of five experiments on such resuscitation of ciliary activity with the aid of a HME are shown in Fig. 6. The main values of four single observations in the five separate experiments recorded at different time intervals are plotted on the diagram, and for each experiment the points that belong together are connected. The zero line observations were made directly with the naked eye and in some cases also recorded but without any measurable ciliary activity.

TABLE 11 *The rate of ciliary beat of mucous membrane pieces related to the time they have been exposed to artificial ventilation in a tracheal model provided with an inlet constriction and a heat and moisture exchanger.**Figures in Italics are means*

Rabbit no	Time after death, min	At start	Beats per minute												
			Time of exposure to artificial ventilation, minutes												
			5	10	20	25	30	50	60	70	90	100	120	150	
1	80	—	—	—	420	—	441	—	—	—	—	—	—	—	
		—	—	—	418	—	433	—	—	—	—	—	—		
		—	—	—	431	—	417	—	—	—	—	—	—		
		—	—	—	423	—	433	—	—	—	—	—	—		
		—	—	—	423	—	439	—	—	—	—	—	—		
1	240	588	503	471	—	—	—	—	457	—	—	—	410		
		559	516	468	—	—	—	—	432	—	—	—	410		
		589	519	475	—	—	—	—	444	—	—	—	433		
		593	490	451	—	—	—	—	411	—	—	—	431		
		570	507	467	—	—	—	—	444	—	—	—	433		
3	30	413	—	—	—	—	—	—	—	536	—	—	—		
		413	—	—	—	—	—	—	—	503	—	—	—		
		413	—	—	—	—	—	—	—	533	—	—	—		
		413	—	—	—	—	—	—	—	536	—	—	—		
		413	—	—	—	—	—	—	—	522	—	—	—		
4	65	—	—	—	584	580	580	670	—	—	—	—	—		
		—	—	—	616	600	629	632	—	—	—	—	—		
		—	—	—	616	629	610	629	—	—	—	—	—		
		—	—	—	616	629	610	619	—	—	—	—	—		
		—	—	—	608	610	607	631	—	—	—	—	—		
4	135	—	651	638	616	—	—	—	—	—	—	—	411		
		—	651	629	626	—	—	—	—	—	—	—	429		
		—	638	629	661	—	—	—	—	—	—	—	429		
		—	640	629	626	—	—	—	—	—	—	—	450		
		—	645	631	632	—	—	—	—	—	—	—	433		
4	155	616	—	—	—	—	—	—	—	—	—	610	—		
		670	—	—	—	—	—	—	—	—	—	619	—		
		619	—	—	—	—	—	—	—	—	—	619	—		
		670	—	—	—	—	—	—	—	—	—	613	—		
		644	—	—	—	—	—	—	—	—	—	626	—		
III	80	629	—	—	—	—	—	—	—	—	590	—	—		
		637	—	—	—	—	—	—	—	—	629	—	—		
		592	—	—	—	—	—	—	—	—	615	—	—		
		635	—	—	—	—	—	—	—	—	596	—	—		
		623	—	—	—	—	—	—	—	—	613	—	—		
5	105	—	612	—	—	—	—	—	—	—	—	—	—		
		—	629	—	—	—	—	—	—	—	—	—	—		
		—	629	—	—	—	—	—	—	—	—	—	—		
		—	638	—	—	—	—	—	—	—	—	—	—		
		—	627	—	—	—	—	—	—	—	—	—	—		

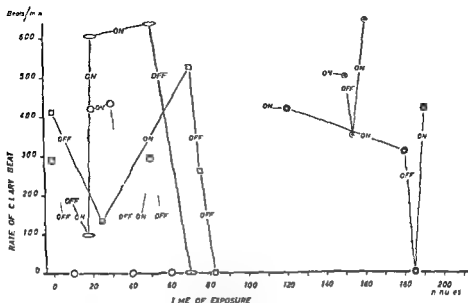


FIG. 6 The rate of ciliary beat in beats per minute related to the time during which the specimens were exposed to artificial ventilation. O — a heat and moisture exchanger was used. □ — the heat and moisture exchanger was disconnected.

DISCUSSION

The purpose of the present investigation was to work out an experimental method with the aid of which it was possible to study objectively the specific influence on the rate of the ciliary beat exerted by different physical factors one by one.

For pure physiological determinations *in vivo* studies are naturally the most reliable ones. During such circumstance, however, it may be difficult, not to say impossible, to reproduce entirely controllable and constant conditions as regards, for example, ventilation, air flow patterns, degree of wall contact between the mucous membranes and the respired air etc. as there are individual anatomical as well as physiological variations. Furthermore, anesthetics must be used which means introducing pharmacological factors so that it may be difficult to obtain reproducible experimental series.

Therefore it became necessary to develop a new modification of the *in vitro* studies on ciliary activity. The equipment described above, with its regulable but constantly working *in vitro* ventilation in constant temperature and humidity conditions, does not only seem useful but also preferable for testing single physical and also chemical factors in comparative experiments.

In the present cases no pharmacological substances were administered to the animals or the respired air and therefore chemical factors will not be discussed in the following account.

Recently Krueger and Smith showed that different electrically loaded air ions had an effect on the rate of ciliary beat *in vivo* as well as *in vitro*. In the

present experiments however such phenomena need not be considered as the experiments were carried out in almost identical air conditions without any addition of other gases. In the following only such physical factors as effect aerodynamic phenomena will be taken into account.

It is well known that ciliary activity is highly sensitive to drying effects and it was appropriately pointed out by Proetz in 1933 that the only enemy known to the cilia in their line of function is excessive drying. This statement has later on been confirmed by many authors recently listed by Dillmann (1956).

During nasal respiration the tracheal mucosa is preserved from damage due to drying as the inspired air is well conditioned already when it reaches the subglottic space (Ingelstedt and Toremalm 1960b). During oral breathing of some duration in ordinary room air as is often the case with unconscious patients and above all with tracheotomized and laryngectomized ones the tracheal mucous membranes below the cannula are exposed to drying which causes dangerous complications if prophylactic steps are not taken. As shown by Dillmann the rate of ciliary beat ceased already at a relative humidity of 50–70% and as room air usually (at least during indoor conditions in the winter months) is below 50% such clinical complications must be expected to occur.

From other experiments (Toremalm 1960a) it was also obvious that the risk of drying effects on the trachea of tracheotomized patients is very high. On the basis of temperature measurements with the aid of a micro psychrometer it could be calculated that the inspired air must be supplied with about 500 g of water in the state of vapour from the trachea and larynx in order to acquire full saturation at body temperature of the inspired air when it reaches the alveolar level whereas during normal nasal respiration only about 100 g are needed.

In this connection however not only room air conditions but also an above all aerodynamic phenomena must be considered. It was shown by Ingelstedt and Toremalm (1960a) in model experiments that the degree of wall contact between the wall of a tracheal model (22 mm in diameter) and room air intermittently sucked through it was highly influenced by the shape of the inlet of the model because of variations in the aerodynamic boundary layer along the tube wall. Within this layer which is caused by friction forces the air flow is always laminar in spite of central turbulence and the layer has an insulating function which prevents too intense a contact between the wall and the passing air. In the paper mentioned it was also assumed that the same physical phenomenon must be considered during biological conditions. It therefore became necessary to verify this assumption which can easily be done by studying the ciliary activity on mucous membranes with the present method in the same experimental conditions. In addition it became possible to achieve an objective biological test of the efficiency of the HMI recommended by the author for clinical use in post tracheotomy care (Toremalm 1960a, b).

The significance of the method for computation of the present results is thoroughly discussed by Dalhamn (1960). His *in vivo* determinations of the rate of ciliary beat on rabbit tracheas during nasal breathing showed a frequency of about 1100 beats per minute with a variation between different individuals of 172 beats per minute. The experimental error was 40 beats per minute with a film speed of 220 exposures per second. The present studies were made with 64 exposures per second which means that the experimental error must be higher. But this fact does not seem to be of any importance for the present results which are not intended to reflect physiological conditions especially as the trends of the experimental series are so unequivocal. The rate of ciliary beat in the actual series was however, initially only about 600 or about 50% less than in Dalhamn's series. This may be due to excision of the mucosa to the time between death and the beginning of the experiments and also to the fact that filming could not be started until the microscope and the camera were well focused on the specimen. During this periods four of five ventilatory cycles took place.

As seen from the single recordings in Table I and II and in Fig. 5 the relatively dry room air in connection with the flow pattern beyond the constriction had a deleterious influence on the ciliary activity. Within 20-30 min the cilia stopped beating whereas during relatively equal aerodynamic conditions with the use of a HMF the initial activity could be preserved for up to eight hours and possibly even longer especially if lesser tidal volumes had been used. This latter fact makes it possible to study how for example different chemical substances influence the ciliary function during several hours.

As regards the clinical use of HMF's the results obtained in the second experimental series provide evident information. Connected to an ordinary tracheal cannula the HMF is able to preserve ciliary activity and thus normal mucous flow. The latter function has also been verified clinically by the use of a device capable of automatic evacuation of tracheo-bronchial secretions which are carried to the inner end of a tracheal cannula mainly through ciliary activity (Toremalm 1960c). Furthermore the ciliary activity could be reactivated after a short standstill with a HMF.

From the present investigation it can be concluded

1. That the present method for determination of the rate of ciliary beat in a tracheal model during artificial ventilation is suitable for studying the effect on the ciliary activity in the respiratory tract exerted by single physical factors and chemical substances.
2. That aerodynamic phenomena such as air flow patterns, degree of wall contact and type of boundary layer distal to a constriction (larynx, tracheal cannula, tracheal stenosis) have an inhibitory influence on the ciliary function.
3. That it is possible with the aid of HMF's not only to preserve but also to regain ciliary activity which has just before been stopped by drying.

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VERTIGINOUS ATTACKS IN THE LIGHT OF FOLLOW UP STUDIES

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A summary will be found at the end of this article

The etiology of vertigo has been the subject of numerous investigations. As is well known, vertigo may occur as a symptom in various diseases; it may be due to an inner ear lesion as in Meniere's disease, endo- or exogenous toxins, cardiovascular diseases, or to neurological factors.

Heikinheimo (1956) published a report based on case records of the patients admitted to the Provincial Hospital of Turku for acute vertigo. It was the purpose of his study to throw additional light on the symptoms of these patients and the factors underlying their vertigo. His material consisted of 100 patients from the period 1949-1954. Those over 30 years old showed no differences in regard to age and sex; in patients under 30, paroxysmal attacks of vertigo were fairly rare. About 50 per cent of the patients were admitted because of a first attack, while half of the patients had had several attacks before. The most frequent associated symptoms were nausea and vomiting, headache, spontaneous nystagmus, positive Romberg test, and hearing loss. In dealing with the etiology, Heikinheimo arrived at the conclusion that an organic vestibular lesion was responsible for the vertigo in 53 cases. Fifteen of these 53 cases were recorded as definite Meniere's disease on the basis of recruitment demonstrated audiometrically and of a more profound hearing loss for low tones. The age and sex distribution of these patients was similar to that of the entire material, but in this group spontaneous nystagmus and tinnitus were about twice as frequent as in the other patients. In the rest, i.e. in about one half of the cases, there were no lesions of the vestibular apparatus, nor were any other factors disclosed to account for their vertigo.

The present work is based on re-examination of the patients hospitalized for acute vertigo after a follow-up period of several years. The aim was to find out, as far as possible, whether after such an interval new symptoms had been added to the picture or the previous symptoms had become more clearly defined, whether diagnosis could now be established, and in particular whether previous cases of indeterminate vertigo had developed into definite Meniere's disease. The patients who were re-examined consisted of those treated for vertigo in the Medical Clinic and referred to the Otolaryngological Clinic in 1949-1956 for examination of the vestibular system. These patients totalled 127 according to the files of the Otolaryngological Clinic. An inquiry as to the presence of vertigo was sent to all these patients, requesting them to come for follow-up. Seventy-two replied to the questionnaire and 43 presented themselves for examination. These latter underwent a complete

otorhinolaryngological examination including audiograms tests for recruitment (Fowler) caloric tests (Hallpike) corneal sensitivity coordinated movements postural nystagmus Barany's pointing test Romberg and blind fold walking

The age and sex classification of the patients is given in Table 1. The present series shows a slight female preponderance and differs in this respect from that reported by Heikinheimo.

The earlier diagnoses of the patients appear in Table 2. Diagnosis was Ménière's disease in 17 cases vertigo in 12 the following seven diagnoses were about evenly distributed among the remaining 14 cases: neurocirculatory dystonia cephalalgia neurasthenia cochlear degeneration degeneration of the VIIIth cranial and vestibular nerve acute infection and hypertension. The diagnosis of Ménière's disease was queried in 3 cases being evidently based only on the patient's history. Of these three patients none showed changes in hearing or in caloric sensitivity and none had a positive recruitment test. The remaining 14 showed some objective signs or signs but the number of unmistakable Ménière cases with the typical history and signs was only five. The diagnosis vertigo had evidently been used when the giddiness was extremely strong but the patient could not be put in the Ménière group because the history was incomplete and symptoms were few or slight. A case in point is Mrs. F. M. aged 44 who on admission showed spontaneous nystagmus of third degree to the left hearing being bilaterally depressed but no recruitment could be demonstrated by Fowler's method and the caloric test with water (20°C) revealed no abnormality. The other previous diagnoses need scarcely be dealt with in greater detail here.

In the present study the patients were divided into three groups on the basis of the findings: Ménière's disease cochlear degeneration and vertigo. Table 3 shows the number of patients and the frequency and nature of vertigo in each group. If the patients had paroxysmal attacks of vertigo at least twice a month vertigo was considered to be frequent; if such attacks occurred at least twice in half a year it was considered rare and if more seldom still the patients were put in the group very rare. According to type the vertigo was divided into two groups only: rotatory vertigo and other vertigo (this latter including all other types of giddiness). The follow up study still consisted of 19 patients with frequent 12 with rare and 12

TABLE 1 Age and sex classification of patients

Age	Men	Women
21-30	1	~
31-40	1	4
41-50		3
51-60	6	11
Total	15	23

TABLE 2 Previous diagnoses

Diagnosis	Number
Ménière's disease	17
Vertigo	12
Neurocirculatory dystonia	3
Cephalalgia	3
Neurasthenia	3
Cochlear degeneration	9
Degeneration of VIII th cranial and vestibular nerve	1
Acute infection	1
Hypertension	1

with very rare attacks. The table further shows that 13 cases were now diagnosed as Ménière's disease, thus two less than in the earlier study. There were 8 patients with cochlear degeneration and 20 patients were placed in the group of vertigo; these last had no auditory disturbances.

Thus the Ménière group now included only 15 patients against the previous figure 17. It must be added, however, that four patients earlier listed under other diagnoses had now acquired the typical characteristics of Ménière's disease: paroxysmal vertigo, tinnitus, nausea, typical changes in the audiogram and the recruitment phenomenon. On the other hand, six patients previously classified as Ménière's disease were now placed in the group of vertigo because of the few objective symptoms. In these cases hearing was normal and the caloric test showed no pathological features.

The auditory changes in the Ménière group are illustrated in Table 4. The audiometric changes are grouped according to the audiogram types presented by O. Meurman and Grönne (1956). The table shows that the curve was depressed throughout the tone range (125 to 8000 cps) in 7 patients, in one of whom no recruitment was demonstrated by Fowler's test. This is probably explained by the fact that this was a follow-up study and the absence in these cases of a marked recruitment phenomenon. The patient referred to above who showed no recruitment had been ill for 11

TABLE 3 Follow-up of examined patients classified by frequency and nature of vertigo and by diagnosis

Diagnosis	Frequency and nature of vertigo			Total		Total
	Frequent	Rare	Very rare	Rotatory vertigo	Other vertigo	
Ménière's disease	11	—	2	12	3	15
Cochlear degeneration	4	2	2	8	3	8
Vertigo	11	3	8	11	9	20

TABLE 4 *Hearing loss in affected ear of Ménière patients and (if reverse of) recruitment*

Audiogram	Number	Recruitment
Depressed throughout whole range	7	—
Rising	1	—
Slowly falling	2	—
Abruptly falling	1	—
Encastre 1 ear	1	—

years. The average hearing loss in these cases was 40 db in the affected ear. Four patients had a rising audiogram, the rise being on an average from 40 to 10 db. A gradually falling audiogram was recorded in two cases (drop from 30 to 70 db) and an abruptly falling curve in one patient. In this last case the loss was 20 db at 2000 cps but as much as 70 db at 4000 cps. In this patient too recruitment could not be demonstrated with Fowler's test.

As far as the caloric excitability of the Ménière patients is concerned (Table 5) the results are in line with those of Hallpike. Because of the small number of cases in the total series, the distribution into the groups does not appear so clearly as in the case of a larger series. Fig. 1 shows the results of the caloric tests of three Ménière patients. The first patient has a distinct directional preponderance to the side of the healthy left ear and in the right ear the loss is about 50 db. The second patient, with a loss of 30 db throughout the whole range in the right ear, shows a distinct canal paresis on the right side. The third patient, on the other hand, exemplifies a normal caloric reaction in an otherwise typical case of Ménière's disease.

A study of the other two groups revealed neurological abnormalities in only one patient who is dealt with separately below. The caloric sensitivity evaluated by the method of Hallpike was within nearly normal limits when taking the mean ± 22 (standard deviation) to be normal. However, as far as concerns total sensitivity, it is found that the response to caloric stimulation in almost all patients with cochlear degeneration was at the lowest normal limit and in normally hearing persons only slightly above this low limit. Hallpike as is known reports the mean total sensitivity as 120 s and the standard deviation as 60 s. Thus, it seems that 100 s should still be regarded as normal—a result in fact shown by almost all patients in these two groups.

The single patient whose neurological status showed abnormal features was Mrs. M. O., aged 36. She was first admitted in 1931 complaining of

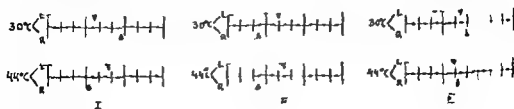


FIG. 1

TABLE II *Caloric sensitivity in Meniere patients*

Directional preponderance to normal healthy side	7
Canal paresis	6
Directional preponderance + canal paresis	3
Normal reaction	3

acute strong vertigo of undetermined nature and she had since been suffering from increasingly severe attacks. Examination showed that blindfold walking was impossible because of great unsteadiness. The reaction to Romberg's test was also severe unsteadiness though there was no distinct falling tendency in any one direction. Hearing was entirely normal. No response to caloric stimulation was elicited even with ice water. The rotation test gave a very indistinct reaction. Bárány's pointing test and co-ordinated movements were normal. The original diagnosis had been degeneration of the VIIIth cranial and vestibular nerve. However this seems to be a case of some kind of toxic reaction in the vestibular apparatus.

It should be kept in mind when evaluating the results obtained that this was a follow up study in which an interval of several years (on an average 6.5 years) had elapsed since the previous examination. Consequently it had been expected that certain symptoms would now have been more distinct than before. However apart from the fact that four new Meniere cases were disclosed (nearly 10 per cent of the total re-examined) it can scarcely be said that the present diagnoses were more definite than the earlier ones. Possibly the vertigo in the cases of cochlear degeneration can be explained on the basis of general degenerative changes in the inner ear and central nervous system (I. J. Ormæ & M. Koskenoja). This seems to be indicated also by the decrease in total sensitivity. Yet nearly half the patients the group of vertigo were found to lack objective findings in spite of the diversity of the history.

SUMMARY

The present paper is a follow up study of 43 patients referred because of vertigo from the Medical to the Ear Clinic in the period 1919-1936. They were re-examined in an attempt to find out whether this interval of several years had brought to light new symptoms or signs or intensified the original symptoms and whether it was now possible to establish a definite diagnosis above all whether cases of indeterminate vertigo had meanwhile developed into unmistakable Meniere's disease. The patients underwent an otorhinolaryngological examination including audiograms Fowles test for recruitment Hallpike's caloric test tests for corneal sensitivity co-ordinated movements positional nystagmus Bárány's pointing test Romberg and blindfold walking. As this was a follow up study the number of old patients exceeded that of the younger. There was a slight female predominance. The original diagnoses had been: Meniere's disease (17) vertigo (12) neurocirculatory dystonia (3) cephalalgia (3) neurasthenia (3) cochlear degeneration (2) degeneration of the VIIIth cranial

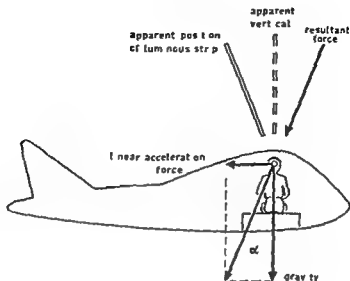


Fig. 1

horizontally with uniform velocity in a straight direction. The test person is facing the left hand side of the plane; the cockpit is dark, and at eye level in front of him is an illuminated vertical strip. If now a linear acceleration with subliminal angular acceleration occurs, the test subject will observe an anti-clockwise rotation of the vertical strip. The rotation is a result of both the gravity and the linear acceleration. The diagram is drawn with regard to the otoliths of the test subject.

A similar situation can be created by a centrifugal force as shown in Fig. 2. Here the test subject rotates in the dark with his sagittal plane perpendicular to the radius and the axes on his left side. Now again the illuminated vertical fixed in front of him will rotate clockwise, this being caused by the resultant of gravity and centrifugal force, angular acceleration being subliminal again.

There is a difference of a mechanical nature between the two cases in regard to the influence of the otoliths on the macula. In the first case the direction of action on the macula is projected on the other side of the vertical as in the latter. Both phenomena are called *oculogravic illusions*.

The utricle is the generally accepted receptor for the new apparent vertical; the function of the saccule in this matter is neglected. Jonckheer, however, proved in animal experiments that in a tempo-temporal acceleration the saccule may be stimulated. This kind of acceleration lies in the same frontal plane as the directions of the above mentioned accelerations. That the receptor function of the oculogravic illusion concerns the labyrinth is shown in the fact that many deaf mutes as well as persons with bilateral non-functioning labyrinths do not perceive this change in position of the vertical.

The explanation of the oculogravic illusions is given by the obviously present tendency to bring the optical and vestibular orientation in agreement.

Also stimulation of the semicircular canals gives rise to an optical illusion.

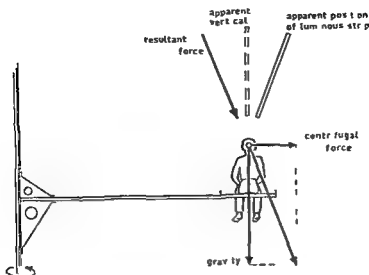


Fig 2

called optogyril illusion. We are not convinced that this illusion remains phenomenologically apart from the oculogravic illusion initiated by stimulation of the otoliths. We are once again reminded of the centrifuge principle. If one applies an angular acceleration supraliminally with regard to the semicircular canals a nystagmus will occur. If a test person rotates in the dark with a light point fixed in front of him on the rotation disc then there occurs an apparent movement of this light point in the direction of the rapid phase of the nystagmus. The movement is a streaming one without any displacement of the visual object. It is possible to demonstrate this phenomenon more simply and more clearly by a caloric stimulation. At the onset the test person sees a horizontally nystagmiform movement of the light point caused by the nystagmus itself; after a short time this movement being replaced by a streaming movement. The direction of this streaming movement is again in the direction of the rapid phase of the nystagmus.

The current theory states that the optogyril illusion is caused by a tracking of the image over the retina. The rapid phase is disregarded while the slow one is perceived whereby a unilateral movement occurs in the direction of the rapid phase. The direction is reversed because there is a ray crossing in the eye (Graybiel *et al.* 1946). Once and for all it must be emphasized that there is a difference between an intended eye movement and eye movements of a reflexive nature or which are externally and mechanically initiated. If one concentrates a light point in the dark during vestibular nystagmus the point is, as already mentioned, displaced as initially flickering in one direction according to the nystagmus. This reflexive eye movement is seemingly not considered in the central calculation concerning the position of the light point in space. If one consciously moves the eyes then one remains orientated as to the position of a visual object in the surrounding space due to the com-

TABLE 1

Nystagmus turning sensation and opto gyral illusion	51%
Nystagmus turning sensation and no illusion	3%
Nystagmus illusion and no sensation	7%
Nystagmus but no illusion and no sensation	36%

bination of sensitive information from the eye muscles and localisation of the image on the retina. This combination is missed in non intended and in reflexive movements. So far the explanation of the opto gyral illusion as stated by Graybiel can be maintained but a displacement of the image over the retina remains obligatory in this theory.

There are however several facts to be reported which do not agree with this concept. Firstly almost every healthy person shows a nystagmus in a caloric stimulation of a semicircular canal. From our experience with 300 test persons all showing nystagmus following caloric stimulation with 300 cc water at 30° irrigated in 30 sec. we have found that only in 17% does a turning sensation occur. Similarly only 61% perceives the opto gyral illusion in the sense of a streaming movement in the direction of the rapid phase of the nystagmus. 54% perceives both phenomena and 16% nothing but nystagmus (Tab. 1). Vegetative symptoms are here neglected.

The global connection that seems to exist between turning sensation and opto gyral illusion was the reason for determining the sensation and the illusion cupulometrically. The cupulogram of a normal test subject in vix shows the diagram of Fig. 3 in accordance with the experiments of Van Dishoeck, Spoor & Nyhoff. Only 18 accurately reproducible cupulograms among 41 normal test subjects are considered. The line concerning the sensation and the line derived from the points of duration of opto gyral illusion run parallelly or coincide. An agreement with the duration of the nystagmus was not found. Similarly the turning sensation shows certain qualities of speed which normally are found again in the velocity of the illusion and not in the velocity of the slow phase of the nystagmus. The discrepancy between the last mentioned velocities can be demonstrated by neutralization of the opto gyral illusion by a real movement of the light point in the opposite direction. We effected this opposite movement by placing the light point on a toy train. By a cord the test subject could manipulate in such a way that the train was apparently at rest. From the distance of the displacement and the time both the average velocity and the average angular velocity can be derived. These qualities of the opto gyral illusion can be compared with electronystagmograms made under the same circumstances. Because of the inhibitory effects of a light point on vestibular nystagmus simultaneous recording of the two phenomena was not possible.

In order to eliminate the tracking of the image point over the retina we have used as a visual object an after image that is projected on the retina in 1/1000 sec. so that no displacement was further possible over the retina during the eye movements. The flash apparatus was fixed on the rotating chair and

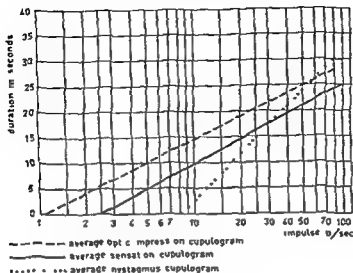


FIG 3

this after image could be initiated just before the stopping of the chair. In order to avoid extrafoveal after images a light point close to the flash apparatus was concentrated during the projection of the after image. This light point went out at the stopping of the chair. Once again one sees a streaming movement of the after image in the direction of the rapid phase of the nystagmus just as with a real visual object, there is movement but no displacement. At the end of the streaming movement the displacement occurs and then there is a lateral position of rest according to the eye deviation. Cupulometry was done again. Besides a larger spreading in the situation of the points and a reduced number of similar cupulograms, another line was found which also follows the sensation line.

The question arises whether it is possible to produce an opto-gyral illusion—an illusion by a stimulation of a semicircular canal—without the presence of nystagmus. Partially this is demonstrated in the lower angular accelerations in cupulometry, and in threshold investigations for the opto-gyral illusion and the turning sensation. These thresholds are lower than those found for the nystagmus (Roggeveen & Nyhoff).

Important clinical observations are reported by Van Dijkhoeck, Spoor & Nyhoff concerning the discrepancy between illusion and nystagmus. In a patient suffering from bilateral abducens paralysis a horizontal nystagmus could not be provoked, whereas the illusion was of normal duration and direction. Moreover, in a patient suffering from monocular total ophthalmoplegia nystagmus could not be provoked in the abnormal eye but was present in the normal eye. The opto-gyral illusion proved to be the same whether the patient observed the light point with his normal eye or with his paralysed eye.

Arguments in favor of the opinion that the opto-gyral illusion does not

depend on nystagmus can be demonstrated also by prolonged caloric stimulation. If one stimulates with water at 40° for 10 min using one ear then there occur during this time turning sensation, illusion and nystagmus. They are however not always present at the same moment for there are phases to consider in which no nystagmus occurs but only turning sensations or only illusion. A continuous course of nystagmus during a stimulation by water at 40° is observed in only some test subjects. There are phases in which the illusion occurs totally independently, the test person seeing the streaming movement of the light point without perceiving turning sensations. The absence of nystagmus is verified by electronystagmography. Moreover already the fixation of the light point is sufficient in most cases to inhibit the nystagmus or to stop it. Use of an after image during prolonged caloric stimulation gives rise to the same experience, however less clearly manifested than in rotation tests.

If one considers that the nystagmus is not the cause of the opto-ocular illusion, the question arises in which direction the cause can be found. We make reference again to the diagrams of oculographic illusion. Here one sees the correlation of a new apparent vertical, thus new information out of the vestibular sphere with a newly connected optical impression.

The opto-ocular illusion is mostly accompanied by a turning sensation. It is at this point that the strongest correlation is supposed. Besides this there is a certain aspect of this correlation which can be illustrated by the diagram in Fig. 4.

In this diagram the test subject is sitting in upright position and facing the axis of rotation. If the centrifugal force approaches the value k , this force results with the gravity g in a resultant force R making the angle α with the vertical. The test subject feels tilted behind in such a way that his longitudinal axis also makes an angle α with the vertical. According to this a light point I_1 fixed in front of the test subject is now perceived in I_2 . By increasing the velocity of uniform rotation till a constant force k can be maintained, this force will result with the gravity in a force R making an angle of α degrees with the vertical. If α is greater than about 40° the light point I_1 is not perceived in the supposed position in I_1 but in I_2 , thus in an angle perpendicular to the floor of the rotation disc. The test person undergoes the sensation of turning with his longitudinal axis parallel to the floor mentioned.

It may be concluded from these facts that in tests concerning the centrifugal principle a correlation between the vestibular and optical perceptions exists below a certain individually critical level of stimulation. Above this level where stimulation is non physiological there is a disorientation. With opto-ocular illusion one has the same experience. A short caloric stimulation may create an almost exact connection between turning sensation and apparent movement. If the stimulation is applied non physiologically as occurs in a continuous caloric irrigation then there is a complete divergence, sensation and illusion can occur independently.

In our opinion these optical illusions must be considered in the following

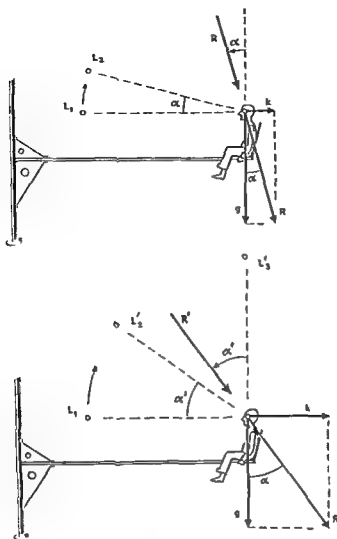


Fig. 4

manner. Orientation is perceived by information from the vestibular, haptic and optical spheres. In daily life they are perfectly in accordance with each other. If one uses non physiological stimulations whereby our information is discrepant, then the association centre does not give false and confusing information to our consciousness, but a correction occurs. The correction is seemingly of such a nature that one piece of information becomes dependent on the other. Maybe on phylogenetic grounds the adaptation takes place in such a way that the optical information is adapted to the vestibular and haptic cues. Together they can be considered as one unit. A visual object must follow the faulty information about the orientation of the vertical and about a non-existent rotation. The result of the adaptation is a non-confusing impression about moving of the subject itself and about localisation of

objects in space in the absence of other optical cues which may contribute to spatial orientation. But also this unit is interpreted wrong and therefore gives rise to an illusion. The corrective power of the hypothetical association centre is restricted. If the stimulations are largely non physiological as by continuous caloric stimulation and in a centrifuge or of more practical importance, under flight conditions then the centre loses its function and apparent connection between the vestibular and optical information totally disappears giving rise to disorientation.

ZUSAMMENFASSUNG

Außer vom Bild auf der Netzhaut wird die Lokalisation eines visuellen Objektes im Raum von Faktoren extra retinaler Natur bestimmt. Verschiedene dieser Faktoren betreffen die Interpretation der beobachteten Phänomene. Jedoch das Labyrinth beeinflusst auch die Wahrnehmung der visuellen Sphäre. Es werden Beispiele von oculografischer und opto-gyrischer Täuschung angeführt, hervorgerufen respektive von der Reizung der Otoliths und von den semizirkularen Kanälen. Im Gegensatz zu der allgemein angenommenen Meinung können Argumente angeführt werden die zeigen daß die opto-gyrischen Täuschungen unabhängig vom Nystagmus sind. Dem Autor gemäß werden beide Arten von Täuschungen als eine Tendenz betrachtet, um die optischen, haptischen und vestibulären Anhaltspunkte in Korrelation zu bringen als ein Ausdruck von zerebraler, korrelativer Funktion. Bei Abwesenheit von optisch orientierender Anhaltspunkte die einen Faktor von Erfahrung beinhalten — was auch der Fall ist bei nur einer Lichtquelle im Dunkeln — soll die vestibuläre Impression dominieren. Innerhalb bestimmter Grenzen ist die Anpassung von der optischen Impression zur vestibulären Information vollständig. Eine stärkere Reizung des vestibulären Apparates verursacht Dissoziation. Eine hohe nicht physiologische Reizung ergibt eine vollständige Abwesenheit von Korrelation was eine Disorientation zur Folge hat, die bis zum Bewußtsein durchdringt.

RÉSUMÉ

Non seulement l'image sur la rétine, détermine la localisation d'un objet visuel dans l'espace mais aussi des facteurs de nature non rétinienne. Plusieurs de ces facteurs concernent l'interprétation des phénomènes observés. Pourtant c'est le labyrinthe aussi qui contribue à la perception visuelle. L'auteur donne des exemples d'illusions oculogyres produites respectivement par la stimulation des otolithes ou par celle des canaux semi-circulaires. Au contraire de l'interprétation régnante on peut produire des arguments montrant que les illusions oculogyres sont indépendantes du nystagmus. L'auteur considère les deux catégories d'illusions comme une tendance à la corrélation des perceptions de nature optique, haptique et vestibulaire étant toutes l'expression de la fonction cérébrale corrélatrice. En cas de manque de perceptions optiques d'orientation — ayant un facteur d'expérience — ce qui est de même avec un simple point lumineux dans l'obscurité — c'est l'impression vestibulaire qui sera dominante. A un certain degré l'adaptation de l'impression optique à l'information vestibulaire est parfaite. Une stimulation plus intense de l'appareil vestibulaire

cause la dissociation Une stimulation grandement non physiologique produit un manque absolu de corrélation et ce manque aboutit à une désorientation pénétrant dans la conscience

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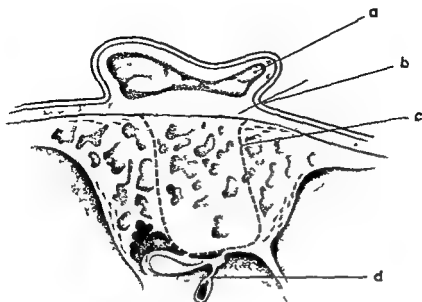


FIG 1 Schematic representation of cross section through operation area (a) Distorted cartilage (b) rudimentary outer ear (c) approx position for incision behind ear (d) projected external meatus (d) ossicles

Session 1 Reconstruction of the auditory canal

First an incision was made behind the rudimentary auricle and the mastoid process was dissected partly free. Next a wide atticotomy was done establishing a new external meatus. This bone meatus was oval in form with the longer axis about 3 cm in length and lying diagonally about 30° from

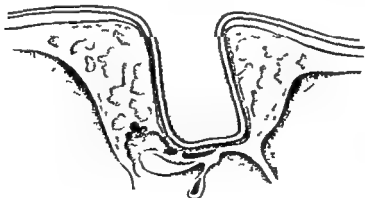


FIG 2 Rudimentary outer ear's skin sac inserted in the meatus

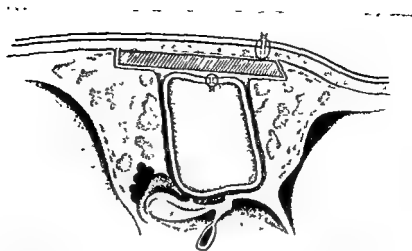


FIG 3 Bone transplant in place with ends inserted in bone recesses. Skin covering for bone transplant is produced from flaps cut from meatus (medial side of the bridge) and from skin immediately outside meatus (lateral side). The sutures of the lateral flaps should be placed in relation to those for medial flaps.

the horizontal (see figures). The cartilage in the rudimentary auricle was then removed, thus producing a thin-walled sac. This sac, which at its formation lies outside, is then pushed in and tamped down to touch the walls, antrum, tympanum and the deformed ossicles. In this way the new meatus is covered with normal skin with good circulation and furthermore membranous contact with the ossicle chain for the conduction of sound has been established. The tamping was continued over a period of about two weeks (Figs 1 and 2)

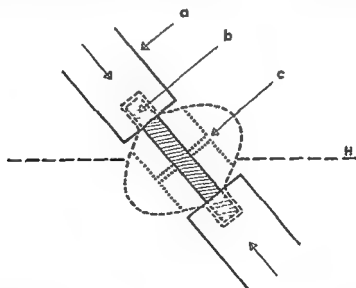


FIG 4 (a) Skin flaps cut from the edge of the oval opening stretch with ease to form skin covering for bone transplant. (b) bone transplant set into recesses in bone. (c) sutures of medial flaps. Horizontal plane (note transplant's direction in relation to this).



FIG 5

FIG 5 Hook made of gold

FIG 6

FIG 6 Hook in place round skin covered bone bridge

Session 2 Establishment of a skin covered bone bridge across the meatus

The skin covering for this bridge on the medial side was achieved in such a way that two flaps of skin of 1.5 cm width were cut out of the meatus and sewn together (Fig 3). A bone transplant from the iliac crest $4 \times 0.5 \times 0.5$ cm was then fitted into recesses cut in the margin of the auditory canal—on one side with undermining. By pushing the ends of the bone under the peristoma a firm hold was achieved. The lateral sides of the transplanted bone were then covered with another two flaps of 1.5 cm width which had been cut immediately outside the meatus (Fig 4). In all our three cases the skin covering of the bone bridge was not difficult because the flaps both in and outside the canal have been remarkably supple and easy to extend.

The bridge lies on the other diagonal from above the top of the mandibular joint backward and down in order to get a hold in thicker bone and also to avoid the joint. The span of the bridge is therefore as short as possible. Because the opening of the meatus is oval the hook by which the prosthesis is fastened to the bridge can be passed more easily round it. The final part of the procedure—the fixing of the prosthetic ear—was usually possible after two months.



FIG 8

FIG 9

FIG 7 Locking pin, hook, and prosthetic ear showing hard acrylic foundation and recesses for hook ends

FIG 8 Hook with locking pin inserted

FIG 9 Prosthetic ear held fast to hook by locking pin

Description of fixing method

A hook was made of gold (Fig. 7) and was then passed round the by now solid covered bridge. One end of this hook has a gold plate which makes good all round contact with the skin itself (Fig. 6). With this hook in place a combined alginate plaster impression is taken of the area to be covered by the prosthetic ear.

A dental plaster cast of the ear area is made. On this cast in which the hook is inserted a thin piece of hard acrylic is made so as to surround both ends of the hook and form a skin tight foundation for the prosthetic ear (Fig. 7). Hook and acrylic piece are then transferred back to the patient and an original of the ear modelled in wax is then fitted *in situ*. The prosthesis itself made of Dacor—a soft acrylic plastic—is then pressed in a plaster piece mould.



Fig. 10 Patient wearing prosthetic ear

The prosthetic ear is locked fast to the hook by means of a metallic peg which is let into a channel in its hard acrylic base and at the same time into holes in the ends of the hook (Figs. 7-9)

The patient with the new ear in place can be seen in Fig. 10

ZUSAMMENFASSUNG

Bei Fällen von rudimentärer Ohrmuschel und Gehörgangsatresie haben die Verfasser den Gehörgang rekonstruiert und nach der im folgenden beschriebenen Methode eine Befestigungsmöglichkeit für eine Ohrenprothese angebracht.

Der Knorpel im Ohr rudiment wurde entfernt und danach die verbleibende Hauttasche in den aufgetrennten Gehörgang eingestülpt (Attico Antrotomie). In einer späteren Séance wurde ein mit Haut bekleideter Knochenspan mit Hilfe eines Knochentransplantates und Hautplastik quer über dem Gehörgang etabliert. Dieser hat die Aufgabe eine Befestigung der Ohrenprothese zu gestatten.

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CLOSURE OF ANTRO ALVEOLAR FISTULAE

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Numerous therapeutic principles and surgical techniques for the closure of antro alveolar fistulae are described in the literature. It is however difficult to make any direct comparison between different methods since only a few large series have hitherto been reported in which the surgical technique has been uniform and the results have been given.

The present series comprises 110 cases of antro alveolar fistulae, operated on by Rehrmann's method from 1955 until the end of September 1960. The main object of the investigation was to check the reliability of this method in a relatively large series by thorough follow up examination. It has been found that when the primary operation has consisted of repair of the fistula by Rehrmann's method the results have been extremely good, i.e. primary healing has been achieved in about 94% of the cases and definite healing in altogether about 99%. It is pointed out that this method is in addition more easily than others combined with radical operation on the maxillary sinus. This is a great advantage in view of the high incidence of coincident sinusitis in the presence of an antro alveolar fistula.

The most common fistula between the oral cavity and the maxillary sinus is the antro alveolar type, i.e. an open communication between sinus and alveolus. Less common are fistulae between the maxillary sinus and the vestibule of the mouth arising e.g. after operation for a tumour of the jaw, a cyst or chronic sinusitis. The present paper deals only with the so called antro alveolar fistulae.

An antro alveolar fistula appears either in direct association with a tooth extraction or as a complication of it, e.g. in an attempt to extract root remnants or curettage of a granuloma of the root apex.

Treatment of these fistulae has always involved multiple problems. Many therapeutic principles and surgical techniques are described in the literature indicating that no definite absolutely satisfactory method exists. Since the methods of operation are so numerous and their modifications often inappreciable the following review is confined to the essential principles of some of the most usual ones.

A simple frequently used method is that described by Zange. After dissecting the gingival mucosa free from the buccal and palatal alveolar wall

the margin of the alveolus is revised so that the gingival mucosa can be sutured without tension. The drawback of this method is that the actual suture line lies directly over the opening of the fistula with resulting poorer prospects of healing.

The commonest principle for closure of a fistula is to mobilize a flap from the surrounding soft tissues: it is then placed over the perforation and sutured to its margins. This can be done by using a palatal flap (Wassmund 1919, Pichler & Trauner 1948) a flap of buccal mucosa (Axhausen 1930) or a mucosal bridge from the palate to the buccal over the alveolar process (Kazanjian & Converse 1949, Schuchardt 1953).

Rehrmann (1936) uses a buccal trapezoid flap of mucosa and periosteum. This technique avoids such drawbacks as granulation formation and difficulties in mobilizing and fitting the flap as in Pichler & Trauner's method as well as the risk of necrosis with Axhausen's flap of buccal mucosa. Finally, one is not—as in the method of Kazanjian & Converse—dependent on the teeth being lacking at the side of the fistula.

Since 1953 Rehrmann's method has been used consistently for closure of intro-alveolar fistulae at the Ear, Nose and Throat Department of Karolinska sjukhuset as it is considered to be superior to many other methods. That this method can more easily than others be combined with radical operation on the maxillary sinus is a great advantage in view of the fact that there is coincident sinusitis in most cases of fistula.

General Principles of Treatment

On broad lines the principles generally applied in treating an intro-alveolar fistula are as follows:

When the fistula is fresh spontaneous healing can be promoted by protecting the blood clot in the alveolus by simple suture of the gingiva. Spontaneous healing takes place by organization of the clot which then becomes covered by ciliated epithelium from the maxillary sinus and squamous epithelium from the buccal cavity. Certain unfavourable circumstances may prevent such spontaneous healing and epithelialization of the fistula then causes it to become permanent—according to Hargrove (1955) as early as within a few weeks. Examples of such unfavourable circumstances are a large perforation ($> 3-5$ mm) an extensive defect in the surrounding gingiva infection of the root apex foreign bodies in the alveolus and in particular complicating sinusitis (Schuchardt 1939, Boenninghaus 1956).

If after extraction of a tooth a root remnant is present in the maxillary sinus the sinus must be opened as soon as possible at the canine fossa and the remnant removed (e.g. Hargrove, Schuchardt). This is to avoid infection (Lindénbaum 1934, Martinsson 1952).

If the root remnant lies in the upper part of the alveolus or has been forced over its edge into the floor of the maxillary sinus without injury to the sinus mucosa it can be removed through the buccal alveolar wall (Boenninghaus).

If however there is the slightest suspicion of lesions of the sinus mucosa the sinus should be opened at the canine fossa for extraction of the remnant and concurrent inspection of the mucosa.

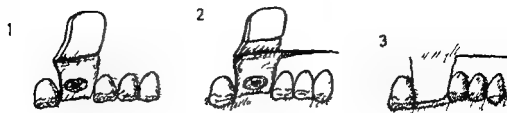
Once sinusitis has developed there is little hope of spontaneous healing of the fistula. Irrigation of the sinus may, however occasionally produce healing and closure of the fistula (Mårtensson 1952 Reading *et al.* 1955). The question is how long this conservative treatment should be continued. It must naturally depend chiefly on the size of the fistula, the appearance of the fluid removed by irrigation and the patient's discomfort. Generally speaking if healing does not take place after 3-4 weeks of such treatment operation is indicated. Endonasal trephination or radical operation on the maxillary sinus then leads to healing and closure of the fistula in many cases.

In a series of 61 cases of antro-alveolar fistulae with coincident sinusitis one of us obtained healing in 4 cases after conservative treatment, in 11 after endonasal trephination and in 46 of the remaining 54 cases after radical operation on the maxillary sinus (Mårtensson). In the other 14 cases i.e. in about a quarter plastic repair of the fistula was necessary. Reading (1955) reported a similar series of 37 cases in 17 of which—i.e. about a third—radical operation on the maxillary sinus did not result in healing. It can be inferred from these figures that the sinusitis clears up and the fistula closes in the majority of cases after conservative and operative treatment. But in at least a quarter of all cases such treatment fails to bring about closure of the fistula thus necessitating a further operation i.e. plastic repair.

It is a considerable disadvantage to be obliged to perform a new operation after a relatively long interval. Moreover the possibility of creating a satisfactory buccal flap has been lessened by the previous Caldwell-Luc operation. Both these drawbacks are avoided by Rehrmann's method in which radical operation on the maxillary sinus can easily be combined with plastic repair of the fistula in the same session.

Surgical Technique

The first step is to make buccal to the fistula two divergent incisions, towards the reflexion of the mucous membrane in the vestibule of the mouth they are made through both mucosa and periosteum. This provides a trapezoid flap of mucosa and periosteum which is dissected free from the buccal alveolar wall and the lateral wall of the maxillary sinus (Fig. 1). The lateral sinus wall is exposed by a horizontal prolongation of the incision, the wall is then opened via the canine fossa in the usual way and a radical Caldwell-Luc operation can be performed. After revision of the fistula (the sinus can also be opened from this site) the flap is lengthened by cutting through the periosteum parallel to the base (Fig. 2). This lengthens the flap by at least 1 cm and allows it to be fitted without tension over the palatal margin of the fistula with a relatively broad contiguous surface (Fig. 3). To ensure optimal conditions for healing in of the flap the epithelium is then excised 3-4 mm



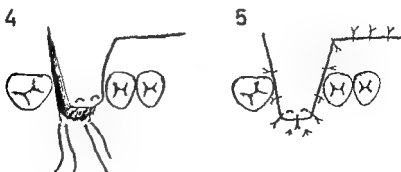
FIGS 1-3 The flap technique

palatal to the fistula. Thereafter we use the suture technique described by Schrøddé (1956). It consists of placing—beside the isolated sutures—2-3 mattress sutures; they pass from the palatal mucosa through the peristoma of the flap and then back to the palatal mucosa where they are tied as shown in Figs 4 and 5.

An objection that has been raised to Rehmann's method is that the vestibule is flattened by the flap so that it is difficult to fit dentures, if required. At Rehmann's clinic it has been shown by careful studies of models after alginate impressions of the vestibule before and after operation that the flattening of the vestibule usually present 2-3 weeks after operation has disappeared after about 8 weeks (Schrøddé 1956). Thus according to Schrøddé one of the causes of the flattening of the vestibule claimed is that the observation period has been too short. The other cause is presumably faulty surgical technique. It cannot in fact be sufficiently stressed that the flap at the base must consist only of submucosa and mucosa so that the soft tissues of the cheek are not displaced when the flap is placed over the fistula.

Present Series

From the beginning of 1955 up to the end of September 1960 114 cases of antroalveolar fistula have been operated on by Rehmann's method at the Ear, Nose and Throat Department, Karolinska sjukhuset. In 110 cases at least one follow-up examination was made in the out-patient department, usually about two weeks after operation. The 4 cases in which data on post-



FIGS 4-5 The suture technique

TABLE 1 Age and sex distribution

Age group years	Women	Men	Total
11-20	5	1	6
21-30	7	18	25
31-40	13	23	36
41-50	8	18	26
51-60	6	5	11
> 60	4	1	5
Total	43	66	109

operative control are lacking are not included in the following discussion. In all 4 cases healing was however fully satisfactory on discharge from hospital 7 days after operation. The cases which on examination in the out-patient department at least 10 days after operation were stated to be primarily healed have been regarded as definitely healed. This is based on the observation of several authors that necrosis does not take place after this time owing to the good blood supply in the flap (Schuchardt 1979, Schrudde 1950, Thullen 1960 and others). This is also in good agreement with our observations since not a single one of our patients denoted as primarily healed has returned with a recurrence, all residual fistulae having been detected during the first 10 postoperative days.

The age and sex distribution in our series are shown in Table 1. It is seen that more than 50% of the patients were 30-50 years old and only 6 were less than 20 years of age. Only one of these 6 was less than 18 years old (12 years). Few cases of antro-alarveolar fistulae in children are in fact described in the literature. This is partly because their maxillary sinus is relatively small as compared with that in adults and is separated from the tooth roots by a thick wall of bone (Hargrove 1955, Reading *et al.* 1955).

As far as the sex distribution is concerned there is a large preponderance of men, particularly in the group 20-30 years. This has also been found to apply in dentogenic sinusitis (Mårtensson 1952) and can possibly be ex-

TABLE 2 Distribution of fistulae caused by extraction of different teeth

Tooth	Total	Percentage
4	2	2
5	13	13
6	60	54
7	31	28
8	4	4
Total	110	100

TABLE 1 *Relation between duration of fistula presence of sinusitis and of root remnant*

Duration of fistula	Sinusitis		No sinusitis		Total
	Root remnant	No root remnant	Root remnant	No root remnant	
Unknown	0	3	0	0	3
<3 days	7	2	7	0	16
3-30 days	11	15	11	1	28
1-12 months	2	10	0	6	18
>12 months	11	8	0	4	14
Total	22	38	9	11	110
	60		20		

plained by the fact that women generally look after their teeth more rationally than men.

The distribution of the fistulae according to the teeth responsible is of some interest and is shown in Table 2. It is seen that more than half (54%) appeared after extraction of the 1st molar and more than a quarter (28%) after extraction of the 2nd molar. These figures are in good conformity with those in earlier series (e.g. Boenninghaus, Mörtensson, Redding, Wasmund).

Von Bonsdorff (1925) and Prieto (1939) have shown that the root apex of both the 1st and 2nd molars is in closest relation to the maxillary sinus. Since the thickness of bone between the sinus and the root apex of the respective teeth is of decisive importance for the occurrence of a fistula, the distribution of the fistulae in the present series according to the teeth is in fact what could be expected in view of the anatomic conditions. In many cases the bony wall over the root apex may be lacking primarily, so that the root membrane lies in direct contact with the sinus mucosa. In other cases the bony wall may have been destroyed by apical osteitis, which has been an indication for tooth extraction.

On extraction of a premolar or molar in the upper jaw a root may be fractured. Attempts to extract these root remnants are apt to force them into the maxillary sinus. As mentioned earlier, such a remnant—which is usually infected—rapidly produces sinusitis. Consequently, it should be removed at the earliest opportunity through an incision in the canine fossa and the sinus mucosa inspected at the same time. If sinusitis has already developed, plastic repair of the fistula is combined with endonasal trephination or radical Caldwell-Luc operation, depending on the degree of mucosal lesions.

In the present series a root remnant was present in no less than 31 of the 110 cases (Table 1). It is further evident from this table how the presence of root remnants affects the incidence of sinusitis and also that this is dependent on the length of time for which the fistula has existed, i.e. on the interval between tooth extraction and plastic repair of the fistula.

TABLE 4 Results of operation

Form of operation	No previous plastic operation		Previous plastic operation		Total
	Primarily healed	Not primarily healed	Primarily healed	Not primarily healed	
Rehrmann	5	0	0	4	9
Endo treph + Rehrmann	18	1	1	0	20
Galdwell Luc + Rehrmann	71	6	3	1	81
Total	94	7	4	5	110

Sinusitis was present in 22 of the 31 patients with root remnants. Seven of the 9 patients without sinusitis had been operated on within 3 days and the other two after 4 and 5 days respectively. This lends support to the view that a root remnant soon gives rise to sinusitis and should therefore be removed without delay. It can be seen that a root remnant was present in 14 of the 16 patients operated on in the acute stage i.e. within 3 days of extraction. In the two remaining cases the perforation was so large that spontaneous healing could not be anticipated. These two were the only acute fistulae operated on apart from those with coincident root remnants. This is probably to be ascribed to the fact that most uncomplicated fistulae heal spontaneously. Consequently they are not referred by the dental surgeon who often closes the fistula himself in the acute stage by simple suture of the gingiva.

The great majority of our cases (62 of 110 i.e. about 60%) nevertheless consisted of fistulae that had been present for more than one month and which had become permanent by epithelialization.

The effect of the time factor on the incidence of sinusitis can also be inferred from Table 3. Thus as early as within 3 days 9 of 10 cases (i.e. more than half) had become complicated by sinusitis. After 3 days sinusitis was absent in only 13 patients in the whole series and 4 of these 13 had previously undergone radical operation on the maxillary sinus. It can be mentioned as a comparison that in Wassmund's series sinusitis was present in 10% of the cases after the 4th day and in 80% after the 7th day.

Thus in our series most of the patients had coincident sinusitis. In view of this fact it is obviously a great advantage that plastic repair of the fistula by Rehrmann's method can easily be combined with radical operation on the maxillary sinus. This was in fact done in the great majority of our cases as can be seen from Table 4 which also shows the results with respect to healing.

Our case material has been divided into two groups i.e. patients who had not undergone earlier plastic repair and those who had been operated on previously with another method. Repair of the fistula by Rehrmann's method was combined with endo-nasal trephination in 20 cases and with radical operation on the maxillary sinus in 81. In 9 cases Rehrmann's operation alone was performed. Of these 9 the 5 in the first group (no earlier

operation) consisted of cases of an acute fistula with root remnants that had been removed through an opening in the crural fossa and of cases in which the sinus mucosa had been inspected through this opening and found to be without pathologic changes. The remaining 4 patients belonged to the second group (earlier plastic repair) and had also all undergone previous Caldwell Luc operation so that only plastic repair was required.

In Table 4 the cases are classified as primarily healed and not primarily healed respectively. By primarily healed we mean—as stated earlier—that healing of the fistula had been observed at follow up examination in the out patient department at least 10 days after operation. Such cases can, as we have already pointed out, be regarded as definitely healed.

Primary healing was achieved in 94 of the 101 patients who had not undergone earlier plastic repair. In 2 of the other 7 cases secondary healing took place without further measures, 2 healed after secondary suture and 2 after renewed plastic repair of the fistula. In the remaining case the fistula did not become completely closed despite a reoperation. Thus in this group definite healing was achieved in altogether 100 of 101 cases.

It is seen that in the second group comprising 9 patients who had undergone previous plastic repair primary healing took place in only 4 cases. In the remaining 5 cases secondary healing occurred in 1 without further measures and in 1 after reoperation, whereas in the other 3 cases the fistula did not close. These 3 patients had undergone more than three previous plastic operations. Earlier plastic repair and a radical Caldwell Luc operation—with customary incision of the mucosa and periosteum in the reflexions of the mucosa in the vestibule—made the flap scarred and difficult to mobilize which may account for the poorer results in this group. The question is in fact whether it would not be advisable in such cases to use a palatal flap according to Pichler & Truner despite the drawbacks pointed out earlier. This would have the advantage of making one independent of the vestibular mucosa that has been scarred by previous operations.

Finally, if the results are considered after combining the two groups primary healing was achieved in 98 cases and secondary healing with or without further intervention in an additional 8 cases, i.e. definite healing in 106 of 110 cases.

ZUSAMMENFASSUNG

In der Literatur sind viele Behandlungsprinzipien und Operationsmethoden für den Verschluss der antro alveolaren Fisteln angegeben. Es ist jedoch schwer einen direkten Vergleich zwischen den verschiedenen Methoden zu stellen, da nur sehr wenige grosse und einheitlich operierte Krankengüter mit Heilungsergebnisse früher bekannt gemacht worden sind. Das vorliegende Krankengut besteht aus 110 antro alveolaren Fisteln, die nach der Methode Rehrmann während der Zeit 1955–September 1960 operiert worden sind. Die hauptsächliche Absicht dieser Untersuchung war an einem relativ grossen Krankengut durch sorgfältige Nachuntersuchung die Zuverlässigkeit der Rehrmannschen Methode festzustellen. Wir haben gefunden

dass wo die Rehrmann Plastik als primäre Operations Methode verwendet wurde waren die Resultate ausserordentlich gut — zirka 94 % Primärheilung und 99 % endgültige Heilung Ausserdem kann diese Plastik leichter als andere mit einer radikalen Kieferhöhlenoperation kombiniert werden, was auf Grund der hohen Sinusitis Frequenz bei den antroalveolaren Fisteln als ein grosser Vorteil angesehen werden muss

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NYSTAGMOGRAPHIC RECORDING OF VERTICAL EYE MOVEMENTS

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The spontaneous position taken up by the eyeballs after closure of the lids has been studied in 50 normal subjects

In 100% of the cases there was initially an elevation of the eyes which however, after some seconds was followed by depression. Subsequently there was reflex activity varying from person to person but not from occasion to occasion in the same person. In 80% of the subjects vertical nystagmus upwards was observed in 6% the vertical nystagmus was directed downwards and in 14% there was no nystagmus but instead vertically undulating eye movements

On these grounds the practice of recording vestibularly induced vertical nystagmus behind closed lids is deprecated

The position taken up by the eyes when the lids are closed was studied by Bell some 150 years ago. He stated that the upward position was natural not only during sleep but also in conscious persons with closed eyes in darkness. Over the past century a variety of opinions have been expressed with regard to the position of the eyes during sleep. Investigating this matter systematically Raehimann & Witkowski (1877) found that the eyes could occupy any position during sleep.

Several workers have interested themselves in the question whether the assumed eye position of rest necessitates sleep or merely results from the act of closing the lids. Most of them arrived at conflicting results in stating that there are considerable differences in the position of rest (Willbrand & Singer 1900, Flischer 1903, Oppenheim 1908, Fuchs 1917, Kestenbaum 1923, Abraham 1931, Weiss 1931). Whilst most of these authors studied patients with facial palsy Hall (1936) made a very comprehensive investigation on normals. Using cinematographic recording of the behaviour of the eyeballs during and after closure of the lids he discovered that the eyes always assumed the same position of rest in the same person but that this position might differ from one person to another. Most often (about 88%) the eyes went upwards and remained there immobile until the lids were opened. In some cases the eyes did not move upwards and in some they actually went down.

The present investigation had a twofold aim. Firstly we deemed it in

interesting to study with modern electronystagmographic methods the behaviour of the eyes behind closed lids. Secondly we desired to establish whether such recording was suitable for registering vestibularly induced vertical eye movements.

METHOD

Fifty members of the hospital staff with normal eye movements and no history of vestibular disturbances were selected for the trial. The electrode for recording horizontal eye movements was placed in the outer canthus of each eye; those for recording vertical eye movements above and below the eye on the vertical line through the pupil with the eye in forward gaze. The reference electrode was placed in one of the auricles. The recorder was a four channel nystagmograph (Schwarzer).

The test subject was initially asked to look at a point marked on the wall. Then whilst the position and movements of the eyes were recorded the subject was requested to close the eyes after 20 seconds and to open them again after another 60 seconds. To ascertain whether any resulting eye movements might be due to a fortuitous position of the bulb above or below the horizontal plane the subject was asked to elevate and depress the eyes behind closed lids for some seconds. The effect of the otolith organs upon the eye movements was checked by having the subject shift his head to different positions.

In one subject unilateral and then bilateral facial palsy was induced by means of xylocain anesthesia near the stylomastoid foramen. Concomitantly eye movements were recorded during closure and attempted closure of the lids.

One subject was given 0.8 g of meprobamat¹ orally one hour before eye movements behind closed lids were recorded nystagmographically.

RESULTS

When looking at a fixed point none of the test subjects exhibited any signs of spontaneous horizontal or vertical nystagmus. After closure of the lids all initially showed an upward deviation of the eyes. Some 3 to 5 seconds later, however, the curves went down below the zero line (Fig. 1a), simultaneously, or shortly thereafter there commenced an ocular reflex activity of somewhat varying appearance in the nystagmograms. In 40 cases (80%) there was a distinct vertical nystagmus upwards of varying amplitude and frequency. In most of these cases the eye movements were composed of two frequencies, viz. a very high frequency of 8 to 10 strokes per second with a considerably slower one ranging from 10 to 30 strokes per 10 seconds superimposed on it (Fig. 1b). The fact that these movements actually were genuine nystagmus, i.e. a repetitive phenomenon with one slow and one rapid phase was checked in doubtful cases by tripling the paper speed from 1 cm per second to 3 cm per second.

¹ = methyl 2-propylpropionyl 1-1-3-3-carbamate acid ester

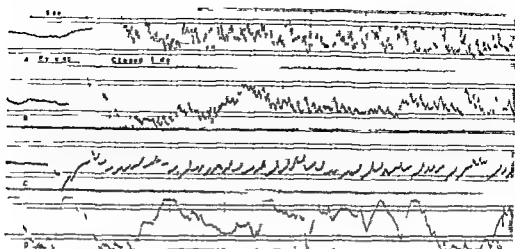


FIG. 1. Types of nystagmograms behind closed lids: A + B fast vertical nystagmus upwards C vertical nystagmus downwards D undulating eye movements

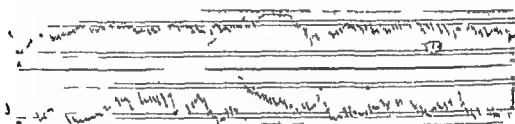


FIG. 2. Nystagmograms of vertical nystagmus behind closed lids with different eye positions: A elevation B depression

Vertical nystagmus downwards was obtained in three cases (6%) (Fig. 1c). Rather than nystagmoid eye movements seven subjects exhibited undulating movements of great amplitude (Fig. 1d).

The nystagmoid activity commencing after closure of the lids continued unchanged for the entire 60 seconds the eyes remained closed. However in two cases it diminished gradually and was replaced by slow undulating movements. Three subjects with particularly marked vertical nystagmus were made to lie down with closed lids for 10 minutes but the recorded nystagmogram remained unchanged in appearance.

Five subjects whose nystagmograms after closure of the lids exhibited dissimilar characteristics underwent as many as six follow up examinations. Yet all of them invariably presented the same highly personal activity pattern.

When the test subjects were asked to elevate and depress the eyes behind closed lids it appeared that the position of the eyes above or below the horizontal plane exerted no qualitative effect on the appearance of the vertical nystagmogram although it occasionally might show minor quantitative changes in the form of reduced amplitude and/or frequency variations (Fig. 2).

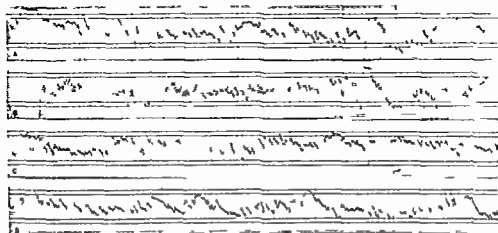


FIG. 3. Nystagmograms of vertical nystagmus behind closed lids with different head leaning positions: *A* backwards; *B* forwards; *C* to right; *D* to left.

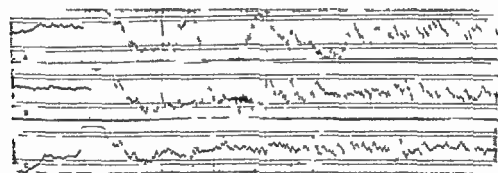


FIG. 4. Nystagmograms of vertical nystagmus behind closed lids in unilateral and bilateral facial palsy: *A* before local anaesthesia; *B* in unilateral facial palsy; *C* in bilateral facial palsy.

The trials designed to elucidate the action of the otolith organs on the appearance of the vertical nystagmogram disclosed that tilting of the head backwards or to either side failed to modify the shape of the curve (Fig. 3).

The test subject in whom unilateral and then bilateral facial palsy was induced showed no qualitative change in the nystagmographic picture. A very slight frequency reduction could be observed already after the induction of unilateral palsy, and it remained sensibly unchanged when both facial nerves had been paralyzed (Fig. 4).

The nystagmogram for the subject given meprobamate presented a recurring phase variation in the form of practically complete interruption of the nystagmus every 10th second or so (Fig. 5).

Although the point is of subsidiary interest in the present context, it may here be mentioned that 11 subjects (22%) showed also horizontal nystagmus eight to the right and three to the left.

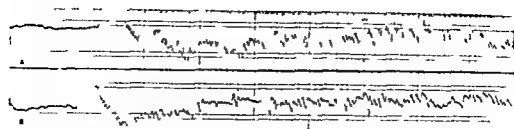


FIG. 7. Nystagmograms of vertical nystagmus following closure of the eyelids. A, left eye; B, right eye.

DISCUSSION

It has long been recognized that the eyes change position after closure of the lids. The consensus of opinion seems to have been that the eyes take up a position of rest which is constant in and peculiar to a particular person. Nevertheless the present investigation has revealed that just as Bell reported a century and a half ago, the eyes went up after closure of the lids in all the examined subjects. But the results deviate slightly from Hall's who was unable to demonstrate an upward movement of the eyelids in 11.6% of his subjects. The latter investigator stated moreover that the eyes remained stationary in their new position which in no case happened in the present investigation. Here it was found that on the contrary the curves after some seconds once again go down below the zero line. The mechanism of the initial elevation has not yet been fully unravelled but it is most likely caused by the reciprocal activation of sets of extraocular muscles. Björkl (1934) electromyographically studied the coordination between antagonistic muscles in patients with facial palsy. He noted a sudden inhibition of activity in levator palpebrae superior when attempts were made to close the eyelids on the affected side. Concomitantly there was a rapid increase of action potentials in the ipsilateral superior rectus and the contralateral orbicularis oculi. Such reciprocity between orbicularis oculi and levator palpebrae superior had previously been demonstrated by Björkl & Kugelberg (1933) and later by Breinin & Moldaver (1935).

An interesting question in this connection has been whether the contraction of the superior rectus following closure of the eyelids is due to synergism with orbicularis oculi or to antagonism towards levator palpebrae superior. Although it remains unclear which alternative is correct the coupling must obviously be very loose and ceases after a few seconds. The fact that the appearance of the nystagmogram remained substantially the same after induction of both unilateral and bilateral facial palsy has been interpreted to mean that the activity of the orbicularis oculi would seem to bear no relationship to the activation of the superior rectus.

It is a highly remarkable fact that closure of the eyelids so frequently produces vertical nystagmus. The same applies to the personally characteristic appearances of the nystagmograms. The phenomenon does not seem

to depend upon voluntary activation or inhibition because elevation and depression of the eyes did not sensibly affect the appearance of the nystagmograms. For the same reason it may perhaps be concluded that the activity level in the muscle spindles arising from the voluntary contraction or relaxation of the superior rectus does not influence the basic reflex pattern. Nor is there anything to suggest that the otolith organs exert any activity capable of triggering this vertical nystagmus. Changes in the position of the head failed to modify the nystagmographic pattern qualitatively.

It seems probable that the nystagmogenic reflex activity demonstrated by the present investigation is mediated via central reflex centres, especially by interaction between the nuclei of the extraocular muscles and the supra-nuclear gaze centres, viz. the reticular formation and the interstitial nucleus of Cajal. This possibility is suggested by the nystagmograms recorded from the subject given meprobamat prior to the trial. The rhythmically varying vertical nystagmus obtained may be interpreted as a constantly fluctuating central activity having repercussions on the peripheral muscular activity. It seems most likely that the phenomenon is due to activity from facilitatory centres alternating with activity from inhibitory centres.

Since 86% of the subjects examined after closure of the lids exhibited some kind of vertical nystagmus, electronystagmographic recording *behind closed lids* must be deemed unsuitable for studying vestibularly induced vertical nystagmus.

ZUSAMMENFASSUNG

Die spontane Position, welche die Augengloben nach Schliessung der Lider angenommen haben, sind bei 50 normalen Personen studiert.

In 100% der Fälle gab es initial eine Elevation der Augen, welche inzwischen nach einigen Sekunden durch eine Depression ersetzt war. Nachfolgend gab es eine Reflexaktivität, die von Person zu Person variierte, aber nicht von Fall zu Fall bei derselben Person. In 80% der Fälle beobachtete man vertikalen Nystagmus nach oben, in 6% war der vertikale Nystagmus nach unten gerichtet und 11% hatte keinen Nystagmus, aber statt dessen vertikalundulierende Augenbewegungen.

Aus diesem Grunde ist die Praxis, vestibular induzierten vertikalen Nystagmus hinter geschlossenen Augen zu messen, nicht zu empfehlen.

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VIBRATORY PATTERN OF THE VOCAL CORDS IN UNILATERAL LARYNGEAL PARALYSIS¹

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Cinematographic roentgenographic and mechanical investigations permit a clarification of the vibratory pattern and the dynamics of phonation in unilateral vocal cord paralysis. The following facts emerge:

- (1) The paralyzed vocal cord vibrates at the same frequency as the normal cord
- (2) The amplitude of the paralyzed cord may be greater or smaller than that of its opposite member
- (3) The motions of the cords may be either in phase or out of phase and may differ substantially from cycle to cycle
- (4) The healthy cord initiates vibration
- (5) The paralyzed vocal cord ceases vibration first

The atypical laryngeal behavior is explained in terms of fundamental anatomic and physiologic principles with reference to structural modifications, air flow characteristics and tissue adjustments. The role of the healthy cord as the regulator of vibration is stressed.

The term unilateral laryngeal paralysis describes a disturbance of the nerve supply on one side of the larynx. Unilateral paralysis may be considered complete when the pathologic process involves the vagus nerve or its central pathways or incomplete when only the recurrent laryngeal nerve is involved. In both cases the vocal cord supplied by the diseased or injured nerve appears immobile on clinical examination: neither abduction or adduction is evident during phonation, coughing or other laryngeal manifestations. Hoarseness generally accompanies this condition, at least during the early stages, indicating an abnormal vibratory function of the vocal cords.

The location of the paralyzed cord varies with the type of paralysis and may range from a median to a lateral position. This diversity has attracted the attention of laryngologists throughout the history of our specialty, resulting in numerous contributions on this elusive subject. Vertical displacement of the paralyzed cord has also been described, although there is no agreement regarding the relative elevations of the two cords. By contrast, little scientific information has been disseminated regarding vibratory movements of the paralyzed cord during phonation. Most clinicians have satisfied themselves

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FIG. 1 Representative photographs of three patients with paralysis of the left vocal cord. Subject 1 demonstrates a complete unilateral paralysis. Subjects 2 and 3 an involvement of the recurrent laryngeal nerve. In each series (a) presents maximum abduction (b) an intermediate position (c) maximum adduction and (d) phonation.

recovery and the left arytenoid cartilage has resumed about 50% of its normal excursion.)

SUBJECT 2—A 67 year old woman whose laryngeal paralysis was associated with cardiac disease. The paralyzed cord was visualized in the paramedian position. The patient usually spoke in a high pitched falsetto with little hoarseness. However, with lowering of the pitch hoarseness and breathiness became quite evident. The pitch range was approximately one octave; the precise limits could not be determined because the lower pitches were scarcely audible above the breath noise. The vocal cords were photographed at a vibratory frequency of 330 cps, near the top of her vocal range.

SUBJECT 3—A 60 year old woman whose laryngeal and vocal symptoms developed gradually over a period of one year. No definite diagnosis had been established at the time of our cinematographic studies but several months later a small malignant tumor was discovered in the left lobe of the thyroid gland. The voice of this patient was weak and intermittently hoarse. The frequency of vibration during the photographic studies was 300 cps.

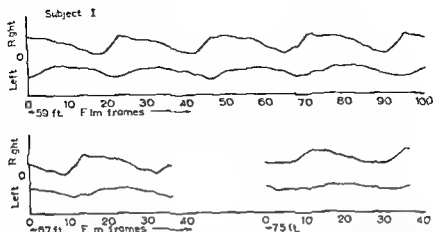


FIG. 2. Analysis of left vocal cord (complete) Subject 1. Graphic representation of vibratory pattern at three successive locations on ultra high speed film during gradual termination of phonation. The upper curve indicates the right and the lower curve the left cord in the excursions from the median (o) line. The horizontal axis depicts the time as measured in film frames.

Findings

The vibratory patterns of the vocal cords in these patients are presented in the accompanying graphs. In all of these drawings the O line represents the median sagittal plane. The upper curve of each pair depicts the lateral and medial movements of the right vocal cord as measured at the center of the glottal margin. The lower curve shows the motion of the corresponding point on the opposite or paralyzed cord. The horizontal axis of the graph indicates consecutive frames of ultra high speed motion picture film.

SUBJECT 1

(1) *Frequency* Fig. 2 shows a typical vibratory pattern for Subject 1. The frequency of vibration is the same for both vocal cords throughout the sequence. This relationship occurs in all films of this subject.

(2) *Amplitude* Fig. 2 also indicates that, in this series, the amplitude of the healthy, right vocal cord is greater than that of the paralyzed cord. In the first complete cycle of the figure, the amplitude of the normal cord is 50% greater than that of the paralyzed cord. Subsequent motion pictures of the same patient, however, indicate a reverse relationship of the amplitude between the two cords (Fig. 3). This finding demonstrates the variety of vibratory motions in the same patient and stresses the need for repeated examination.

(3) *Phase* Perhaps the most striking difference between the curves of the right and left cords in Fig. 2 lies in their respective contours. Inspection of the upper curve reveals a relatively rapid lateral motion of the healthy cord followed by a slower medial excursion. The entire opening movement is accomplished within approximately 20% of the cycle, while the closure occupies the remaining 80%. This proportion is equivalent to a speed quotient¹ of 11/20. The lower curve indicates that the

¹ Speed quotient = Time of abduction i.e. lateral excursion in vibratory cycle divided by time of adduction i.e. medial excursion in vibratory cycle.

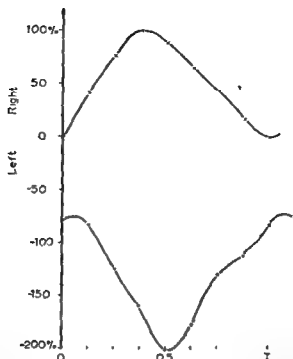


FIG. 3. Paralysis of left vocal cord (complete), Subject 1. Graphic representation of single vibratory cycle from ultra high speed motion picture. The vertical axis shows the amplitude of vibration as a percentage of the normal excursion. The horizontal axis indicates the time (T) of one full cycle, as measured in film frames.

paralyzed cord moves laterally and medially in a symmetrical pattern, with each motion occupying approximately 50% of the cycle (speed quotient = 1.0).

A comparison of the two curves reveals that the lateral and medial motions of the paralyzed cord precede the comparable movements of the normal cord. In the upper pair of curves in Fig. 2, the lateral motion of the left cord begins at frame 11, while the right cord continues to move medially and does not start its lateral excursion until frame 18. This lag of seven frames represents a phase difference of 105 degrees. However, the normal cord reaches its most lateral position only two film frames after the paralyzed cord, a difference of 30 degrees. The medial motions of both cords begin almost simultaneously. Thus, the phase relationship varies throughout the cycle, but the paralyzed cord in this sequence always precedes the motion of its non-paralyzed mate.

In a second series of photographs two months later the phase relationship between the two cords was reversed. The lateral and medial motions of the normal cord preceded similar movements of the paralyzed cord. The amplitude and phase relationships during this second examination are illustrated in Fig. 3. The phase difference in this cycle is approximately 45 degrees.

(4) *Initiation of phonation*. At the initiation of phonation this patient did not expose the vocal cords sufficiently during repeated examinations to permit accurate measurements.

(5) *Termination of phonation*. Changes in the vibratory pattern during the termination of a phonatory episode are illustrated in Fig. 4. The three sections depict three

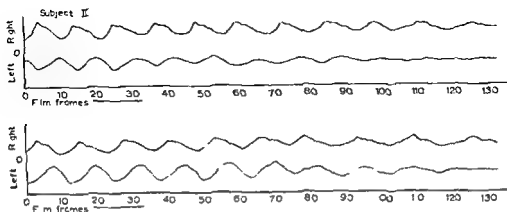


FIG 4 Paralysis of left vocal cord (incomplete) Subject 2 Graphic representation of two phonatory terminations from different ultra high speed films

successive instants covering approximately 17 feet of film (0.22 seconds), during which phonation gradually ceased. This graph demonstrates that the amplitude of the paralyzed cord diminishes more rapidly than that of the healthy cord and that the paralyzed cord comes to a standstill first. The patterns of motion remain essentially the same throughout the entire sequence.

SUBJECT 2

Vibratory patterns of the second patient are indicated in Fig 4. The upper and lower pairs of curves represent different films photographed within a few minutes of each other. Both sections of the figure picture the termination of phonation.

(1) *Frequency* The frequencies of the two cords during the two episodes remain the same. This pattern persists throughout the entire sequence, while the amplitude decreases progressively.

(2) *Amplitude* In the upper pair of curves, the amplitude of the healthy cord is greater than that of the paralyzed cord. However, in the lower sequence, this relationship is reversed: the movement of the left cord is greater than that of the right. The average amplitude of the right cord in the first three cycles of the upper pair is 25% greater than the comparable curves for the left cord. In the lower pair of curves, the reverse relationship holds true, to the same degree.

(3) *Phase* At the beginning of the upper curves in Fig 4, the vibrations of the two cords are in phase, but during progressive cycles this phase relationship changes. After the third cycle asynchrony is readily apparent and is similar to that of Subject 1. At the initiation of lateral motion the paralyzed cord precedes the healthy cord by about 104 degrees. As in the previous patient the lateral excursion of the normal cord occurs more rapidly so that in cycles 4, 5, and 6 the cords are in phase as they begin their medial movements. In the subsequent cycles, each alternation in the direction of motion occurs first on the paralyzed side. This retardation is progressive, so that the phase difference is again 104 degrees by the end of the vibratory sequence.

The lower pair of curves in Fig 4 reveals asynchrony throughout the entire sequence. The start of the lateral motion of the left cord precedes the right by approximately two film frames in the first two cycles, which represents a phase difference of approximately 60 degrees. In the middle of the sequence, the cycle lengthens to

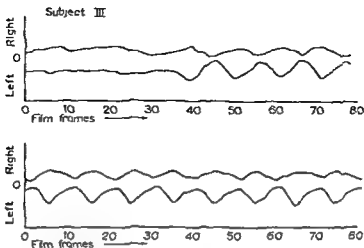


FIG 5 Paralysis of left vocal cord (incomplete), Subject 3. Graphic representation of (a) Initiation of phonation and (b) continued voice production

13 frames and the asynchrony to three frames or to a phase difference of approximately 83 degrees. In the same sequence, the beginning of the medial movement of the normal cord trails the opposite cord consistently by approximately 90 degrees. All movements in the final three cycles are approximately 180 degrees out of phase.

(4) *Initiation of vibration* The exposure of the vocal cords at the initiation of phonation during high speed photography was not adequate for study.

(5) *Termination of phonation* It is evident in Fig 4 that the paralyzed vocal cord stops its vibrations before the healthy cord. This pattern is similar to that of Subject 1 in Fig 2.

SUBJECT 3

Figs 5 and 6 depict representative patterns of vocal cord vibration for the third patient. The upper pair of curves in Fig 5 demonstrates the initiation of phonation, while the lower pair shows a continuing period of phonation. Fig 6 pictures a single cycle for supplementary information.

(1) *Frequency* Throughout the films of Subject 3, both vocal cords vibrate at the same frequency.

(2) *Amplitude* In Fig 5 the amplitude of the paralyzed cord averages twice that of the healthy cord. However, the difference is not typical of this patient, on some sections of film the reverse relationship pertains.

(3) *Phase* The gross pattern of vibration in Fig 5 indicates that the cords are in phase except for an occasional variation of one film frame (36 degrees) at the moment of maximum approximation. The same observation holds true in most other pictures of this patient. However, rarely, the movements of the paralyzed cord precede the normal cord.

The lower section of Fig 5 also illustrates a type of incidental asynchrony which appears frequently in high speed films of unilateral paralysis. The contour of the curve representing the healthy cord changes near the center of the sequence. When the film is projected continuously, the right cord appears to hesitate momentarily and then to settle down again into a regular pattern. During the first 35 frames of

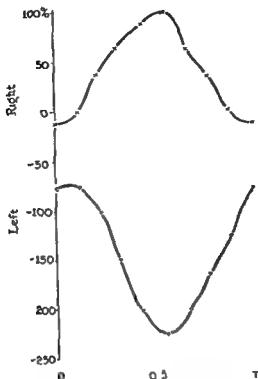


FIG 6 Analysis of left vocal cord (incomplete) Subject 3 Graphic representation of single vibratory cycle from ultra high speed film

this figure, the pattern remains relatively regular thereafter, the right cord pauses momentarily after it has completed one half to two thirds of its medial excursion, and reverses its direction. The subsequent medial movement is interrupted briefly, as shown in the plateau between frames 47 and 49, but the cord continues its motion to its customary medial limit. Following these two aberrant cycles, a normal pattern appears for one cycle and is then succeeded by a single vibration which is 25% longer than those adjacent to it. Subsequently, the prevailing pattern of the curve is re established.

(4) *Initiation of vibration* The upper pair of curves in Fig 5 represents the beginning of one phonation in a rapid series of brief phonatory episodes. The healthy vocal cord starts to vibrate before its paralyzed mate. The small undulations of the normal cord pictured during the first half of this graph had been present for several cycles preceding this portion of the film. During this same initial period, the paralyzed cord remained motionless.

Accurate measurements of cinematographic pictures for illustrative graphs are limited to the lateral and medial movements of the vocal cords. However, these same motion pictures reveal several additional movements which cannot be represented graphically. These movements are particularly striking at the initiation of phonation. First, a series of ripples occurs on the superior surface of the healthy cord preceding the establishment of the regular vibratory pattern. This surface disturbance was not evident on the paralyzed cord. Second, a longitudinal component was observed in both cords, but to a much greater extent on the healthy side. and third,

contrary to the usual impression, the normal cord appeared more pliant than its paralyzed mate

(5) *Termination of phonation* The termination of phonation during high speed photography was not satisfactory for accurate measurements

DISCUSSION

The five basic questions of this investigation have been applied to the findings reported above. The results indicate that

- (1) the healthy and paralyzed cords vibrate at the same frequencies
- (2) the amplitude of the normal cord may be greater or smaller than that of the opposite member
- (3) the motions of the cords may either be in phase progressively out of phase in successive cycles or variably out of phase within a single cycle
- (4) at the initiation of phonation the healthy cord begins to vibrate first and
- (5) at the termination of phonation the motion of the paralyzed cord ceases before that of the normal cord

In short the paralyzed cord vibrates but the pattern of vibration in unilateral paralysis is abnormal

These factual data call for an explanation of the dynamic manifestations in terms of fundamental anatomic and physiologic principles. Three related components stand out clearly as the probable bases of the atypical laryngeal behavior: structural modifications, air flow characteristics and vocal cord adjustments.

The major structural modification in unilateral vocal cord paralysis is the altered position of the involved cord in relation to its healthy mate. Contrary to prevailing opinion, the paralyzed cord does not droop throughout its length but is actually positioned at a higher level than the phonating healthy cord. Recent investigations have produced three fold evidence of this relative elevation of the paralyzed cord.

(1) Roentgenograms of patients with unilateral paralysis of the vocal cords clearly demonstrate this phenomenon (Fig 7). Further roentgenographic evidence may be found in recent articles (Calderon, Cebillos & McGraw 1954; Fink & Hirschner 1959).

(2) The location of the crico-arytenoid joint and the mechanics of its action favor an elevation of the cord in abduction. This phenomenon is demonstrated in the recent investigations of Sonesson (1959) and of the authors (1959). The action of the interarytenoid muscle with its bilateral innervation has a tendency to draw the arytenoid cartilage posteriorly on the involved side thus elevating the vocal process.

(3) This elevation of the paralyzed cord is further demonstrated on our motion pictures. The shadows and relative sharpness of the photographic images in Fig 1 reveal that the paralyzed cords in Subjects 2 and 3 are situated at a higher level than the healthy cords (Moore & von Liden 1960).



FIG 7



FIG 8

FIG 7 Laminagram of patient with paralysis of left vocal cord (complete). Note contrasting contours of cords and elevation of paralyzed member.

FIG 8 Schematic illustration of air flow in unilateral paralysis of the left vocal cord.

The structure of the larynx in unilateral vocal cord paralysis is also modified by the shape of the involved cord. Roentgenograms demonstrate a flattening of the lower lip of the involved cord resulting in an alteration of the typical infra-glottic funnel shape (Fig 7). The contour of the supra-glottic funnel is similarly distorted, though to a lesser extent.

These structural changes modify the characteristics of the air flow in several directions (Fig 8). The funnel at the inferior inlet of the glottis is asymmetrical and tilted, causing the current to be directed at the paralyzed cord and thence through the oblique glottis toward the ventricular fold on the healthy side. The same structural modifications produce a sweeping motion of the air stream against the paralyzed cord during the vibratory cycle. Since the main force of the air current is directed toward the ventricular fold on the healthy side, this structure assumes a prominent part in the vibratory pattern of the larynx. This activity is striking in ultra slow motion pictures, particularly at low pitch. The phenomenon continues throughout the period of vibration. In our experience a complete approximation of the two vocal cords does not occur in unilateral paralysis.

The paralysis of one vocal cord also leads to adjustments within the in-

volved structure. Lack of tonus and atrophy alter the consistency of the soft tissues and decrease the resilience of the diseased cord. These intra cordal adjustments are inversely proportional to the tension of the vocal cords; this factor becomes apparent with changes in frequency. As the vocal cords are elongated with elevation of pitch the atrophic changes are minimized which explains the improvement of voice in different patients.

Review of Cinematographic Observations

In our experience ultra slow motion pictures lead to a more complete understanding of the dynamics of vocal cord vibration than any other single method of investigation. This observation applies especially to unilateral vocal cord paralysis because of the variability of motion from cycle to cycle and the important clues at the initiation and termination of phonation. The rapidity and the transient character of these phenomena prevent their exploration by stroboscopic or roentgenographic means.

In unilateral paralysis the first visible motion at the initiation of phonation is usually an undulation of the ventricular fold on the healthy side. After three or four preliminary excursions of this fold several small ripples traverse the superior surface of the normal vocal cord in a lateral direction. Infinitesimal movements of the free margin accompany this mucosal disturbance. As the healthy cord vibrates with progressively larger excursions the paralyzed cord also starts to move to and fro with a rapid increase in amplitude. In some instances this pattern is interrupted by brief interludes of dissimilar vibrations.

With the establishment of regular patterns of vibration the leading role of the healthy cord becomes evident. The frequency of this cord determines that of its paralyzed mate and the motions of the normal cord regulate phase differences through variations in air flow. The subglottic air pressure displaces the normal cord more readily because of its flexibility and its favored location. Simultaneously the healthy cord deflects the air stream toward the paralyzed cord in a sweeping motion causing a vibratory movement of the paralyzed cord at the same frequency. With each consecutive vibration the shifting direction of the air stream creates a phase difference. The movement of the normal cord toward the center of the larynx, for instance, often results in a lateral displacement of its paralyzed mate. While this phase difference varies greatly from case to case and from cycle to cycle the controlling role of the healthy cord is never open to question. As the phonatory episode subsides the same domination continues; the paralyzed cord ceases its vibration before movement of the healthy cord subsides.

These observations lead us to the conviction that the entire vibratory pattern of both cords is determined by the healthy cord. Current experimentation at our laboratory supports the mechanical and physical aspects of this concept (Moore, von Leden & Smith, 1960).

ZUSAMMENFASSUNG

Kinematographische, röntgenographische und mechanische Untersuchungen ermöglichen eine Klarstellung des Schwingungsmusters und der Dynamik der Lautbildung bei einseitiger Stimmbandlähmung. Die folgenden Tatsachen treten heraus: 1) Das gelähmte Stimmband schwingt mit derselben Frequenz wie das normale. 2) Die Amplitude des gelähmten Stimmbandes kann grösser oder kleiner sein als die seines Gegenstückes. 3) Die Bewegungen der Stimmbänder können in ihrer Phase entweder übereinstimmen oder nicht und können von Periode zu Periode wesentlich voneinander abweichen. 4) Das gesunde Stimmband bringt die Schwingung in Gang. 5) Das gelähmte Stimmband hört zuerst zu schwingen auf. Das atypische laryngale Verhalten wird in Termini der fundamentalen anatomischen und physiologischen Prinzipien erklärt und dabei auf strukturelle Modifikationen, auf die Art der Luftströmungen und Gewebe Anpassungen Rücksicht genommen. Die Rolle des gesunden Stimmbandes als eines Regulators der Schwingungen wird betont.

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VERTIGO AND NYSTAGMUS RESPONSES TO CALORIC STIMULI REPEATED AT SHORT AND LONG INTERVALS

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Experiments on 50 young and from an otoneurological point of view, normal subjects, showed a decline of the average vertigo and nystagmus responses to repeated monaural caloric stimulations with water at 30°C.

This response decline (R.D.) occurred both at short (8 min) and long (24 hours or more) interstimuli intervals which indicates that a central mechanism was responsible for this phenomenon. Statistical analysis disclosed some traits in the response pattern characteristic of habituation.

Of practical interest is the observation that even when an interval of two weeks elapsed between the first and second irrigations the nystagmus response to the second irrigation was significantly weaker. This R.D. was more marked in latency, total number of beats and dysrhythmia than in the duration of the nystagmus response.

INTRODUCTION

A previous investigation (Lidvall 1961) showed that there was a decline in the average vertigo and nystagmus responses to caloric stimuli when repeated at *short intervals* (6 and 10 min) i.e. interstimuli intervals that are only just sufficiently long for one stimulus to cease before the next begins.

The present investigation was undertaken in order to extend the study of the phenomenon of response decline (R.D.) by presenting a larger experimental material which is not only more appropriate for statistical analysis but also makes it possible to compare *short and long intervals* (24 hours or more) i.e. interstimuli intervals that are long enough to exclude the interference of peripheral mechanisms.

MATERIAL AND METHODS

The experiments were performed on 50 healthy subjects between the ages of 20 and 32 years (average 24 years). There were 23 men and 27 women.

Like the subjects who took part in my previous series of experiments, all those who participated in this investigation fulfilled the same requirements as to otoneurological normality and ability to analyse the vertigo responses. Instructions, apparatus and technique of calibration were also the same as those described earlier. Hearing was tested by means of a Quick-check audiometer (manufactured by H. Rappaportbolaget).

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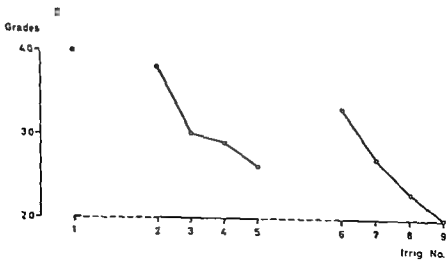
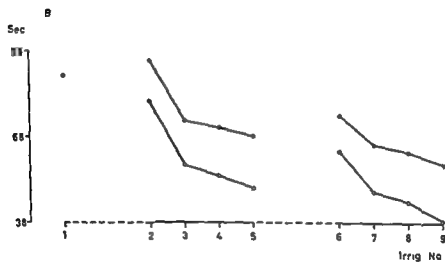
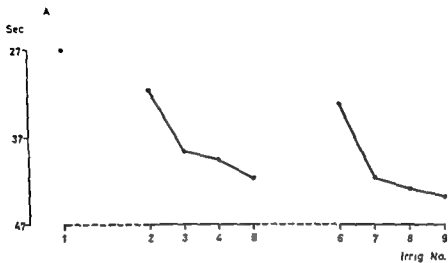


TABLE 1 *Distribution and medians for latency (a), duration (b) and maximum intensity (c) of vertigo responses**n* = number of observations

<i>a</i>									
Irrigation no	1	2	3	4	5	6	7	8	9
Latency (sec)									
0-19	2	11	1	11	1	3	1	1	1
20-39	39	30	20	20	12	25	14	16	13
40-59	8	16	22	21	24	18	22	18	18
60-	0	2	7	9	13	3	13	15	20
<i>n</i>	49	50	50	50	50	49	50	50	50
Median	27.7	32.1	42.8	43.5	46.5	38.1	47.0	47.5	53.5
<i>b</i>									
Irrigation no	1	2	3	4	5	6	7	8	9
Duration (sec)									
0-39	0	3	13	14	17	8	16	16	20
40-79	16	19	27	32	26	30	28	32	27
80-119	27	25	9	3	6	11	6	2	3
120-	11	3	1	1	1	0	0	0	0
<i>n</i>	48	50	50	50	50	49	50	50	50
Median	86.0	81.5	64.5	55.5	52.5	64.0	51.5	51.5	46.5
<i>c</i>									
Irrigation no	1	2	3	4	5	6	7	8	9
Intensity (grades)									
0	0	1	2	4	6	3	7	10	15
1	0	1	5	5	3	1	5	5	4
2	0	2	4	5	14	5	4	8	7
3	4	11	20	18	15	14	17	16	16
4	41	28	16	14	8	21	16	10	8
5	2	10	3	4	4	11	1	1	0
6	0	11	0	0	0	0	11	0	0
<i>n</i>	50	50	50	50	50	50	50	50	50
Median	4.0	4.0	3.2	3.1	2.6	3.6	3.0	2.6	2.4

calculation is that only very few subjects reported a duration of less than 20 sec, which indicates that subjects are usually unable to perceive and/or report vertigo responses of shorter duration than 20 sec. Consequently there is a discontinuity of distribution for values of duration, an inaccuracy that influences the mean values, if "no vertigo" responses are included in the calculations.

TABLE 2 Statistical significance of differences between vertigo responses

n = number of observations \bar{d} = mean difference $s\bar{d}$ = standard error of mean differences *** = highly significant ($0.001 > P$) (P = probability) ** = significant ($0.01 > P > 0.001$) * = almost significant ($0.05 > P > 0.01$)

Difference between irrigations no		2-3	3-4	4-5	6-7	7-8	8-9	1-2	2-6	3-7	4-8	5-9	5-4
Latency 'no vertigo excl													
n		30	30	30	30	30	30	30	30	30	30	30	30
\bar{d}		-7.0	-1.1	-2.2	-8.4	-1.3	-0.8	-4.5	-1.8	-3.2	-3.4	-2.0	8
$s\bar{d}$		1.5	1.1	1.4	1.8	1.6	1.4	1.8	1.5	1.8	1.4	2.1	1
Sign		***	—	—	***	—	—	*	—	—	*	—	*
Duration 'no vertigo excl													
n		29	29	29	29	29	29	29	29	29	29	29	29
\bar{d}		21.0	2.4	3.3	9.9	2.4	4.3	0.8	19.6	8.4	8.4	9.4	-7
$s\bar{d}$		4.6	3.9	3.4	3.5	3.2	2.5	6.0	5.4	4.5	5.6	3.7	4
Sign		***	—	—	**	—	—	—	**	—	—	*	—
Duration all observations													
n		50	50	50	49	50	50	48	49	50	50	50	49
\bar{d}		22.4	3.7	4.3	13.2	3.3	6.6	8.3	17.7	9.3	8.9	11.3	-11
$s\bar{d}$		2.9	2.6	2.7	3.6	3.3	2.5	5.1	3.8	3.2	4.3	3.4	3
Sign		***	—	—	***	—	**	—	***	**	*	**	—
Maximum intensity all observations													
n		50	50	50	50	50	50	50	50	50	50	50	50
No difference		17	21	21	25	22	30	32	23	24	19	21	19
Decrease		10	14	23	22	22	14	8	23	19	25	22	3
Increase		1	10	8	3	6	6	10	4	7	6	7	28
Sign		***	—	**	***	**	—	—	***	*	***	**	***

The number of 'no vertigo' responses increased towards the end of the irrigation series (see Table 1c) and therefore the mean values for all observations may give an exaggerated impression of the R D in the vertigo responses. On the other hand when irrigation series with 'no vertigo' responses were excluded from the calculations (29 series remained) the mean values were too restrictive in this respect. However, as the two curves in Fig. 1b follow essentially the same course it is legitimate to infer that the defect in the accuracy of the evaluation of duration was not of such a magnitude as to distort the type of response pattern. The distribution and medians are shown in Table 1b.

The mean values for maximum intensity (Fig. 1c) were computed from all observations. As the evaluation scale is not equidistant the mean values are inevitably inexact. The distribution of the grades and the medians are shown in Table 1c. The number of rotatory responses (Grades 3 + 4 + 5)

decreased during the course of both short interval series. The medians were on the whole in good agreement with the arithmetical means.

The means of intra individual differences between pairs of values were tested for statistical significance (Table 2). The *t* test was used for latency and duration and the sign test for maximum intensity.

Some features of particular interest will be pointed out here. In both short interval series (2-3 etc. 6-7 etc.) there was initially a highly significant R D in all vertiginous qualities. This indicates that the uniform tendency to R D was always most pronounced at the beginning of the short interval series. An interval of one day brought about a significant or highly significant decrease between some responses of corresponding numbers in the two short interval series (2-6 etc.) in respect of maximum intensity and duration and an almost significant increase in latency. In all qualities of the vertigo response there occurred from the last response in the first short interval series to the first response in the second short interval series (5-6) either a significant or a highly significant increase in the strength of the response.

B. Nystagmus

The mean values for latency, duration, number of beats and dysrhythmia showed a tendency to R D (Fig. 2).

The mean values for latency (Fig. 2a), duration (Fig. 2b) and total number of beats (Fig. 2c) were computed both from all observations and from the observations in all irrigation series except those where Grade 3 dysrhythmia was encountered (32 series remained). These two modes of calculation were employed for obtaining the mean values because when there is a high degree of dysrhythmia considerable inaccuracy in determining latency, duration and number of beats must be expected. Nevertheless except for minor deviations the two curves in Fig. 2a, b and c follow in the main the same course. Hence the inaccuracy involved does not appear to affect the essential trend of the response pattern.

The mean values for the degree of dysrhythmia (Fig. 2d) were computed from all observations. As the evaluation scale is not equidistant the distribution of grades is also presented (Table 3). The number of higher grades (3+2) increased during the course of both short interval series. The medians also increased.

The statistical significance of the mean differences between the pairs of values (all observations, Table 4) was tested in a manner analogous to that used for the vertigo responses.

Some points of special interest. In the short interval series the R D was always most pronounced initially. With an interval of 15 days (1-2) there was a highly significant R D in latency, number of beats and dysrhythmia and an almost significant R D in duration. When the interval between the corresponding stimuli was 1 day there was a highly significant R D in number of beats and dysrhythmia and an almost significant R D in latency and duration.

TABLE 3 *Distribution and medians for grades of nystagmic dysrhythmia*

Irrigation no	1	2	3	4	5	6	7	8	9
Dysrhythmia (grades)									
0	31	17	15	13	11	12	11	8	8
1	19	22	23	18	19	19	13	14	11
2	8	9	7	16	13	13	14	14	16
3	0	2	3	3	7	6	12	14	15
n	50	50	50	50	50	50	50	50	50
Median	0.3	0.9	0.9	1.2	1.2	1.2	1.6	1.7	1.9

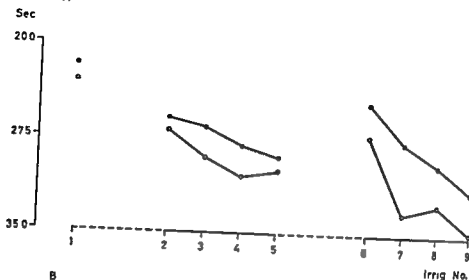
TABLE 4 *Statistical significance of differences between nystagmus responses (all observations)*

Comparison between irrigations no	2-3	3-4	4-5	6-7	7-8	8-9	1-2	2-6	3-7	4-8	5-9
Significance											
n	50	50	50	50	49	48	50	50	50	49	49
d	-2.1	1.6	0.6	-1.4	0.5	-2.1	-3.9	-0.1	-4.4	-1.0	-1.9
st	1.1	1.0	1.0	2.1	1.7	2.0	1.0	1.3	2.2	1.8	2.0
	—	—	—	**	—	—	***	—	—	—	*
Significance											
n	50	50	50	50	50	50	50	50	50	50	50
d	0.0	-0.5	0.1	0.0	6.8	4.5	12.1	3.8	3.9	11.2	10.5
st	3.3	3.1	3.0	3.6	3.8	4.7	5.1	4.4	5.4	6.5	4.9
	**	—	—	**	—	—	*	—	—	—	*
Number of beats											
n	50	50	50	50	50	50	50	50	50	50	50
d	20.6	11.8	5.9	18.4	11.9	7.4	36.1	23.0	21.0	21.1	22.6
st	5.0	4.3	3.7	5.2	3.8	3.0	6.9	6.3	7.0	6.1	5.8
	***	**	—	***	**	*	***	***	**	**	***
Dysrhythmia											
n	50	50	50	50	50	50	50	50	50	50	50
difference	38	35	39	34	31	40	25	25	19	24	26
increase	3	4	2	2	5	3	1	5	5	3	3
decrease	9	11	9	14	11	7	24	20	26	23	21
n	—	—	—	**	—	—	***	**	***	***	***

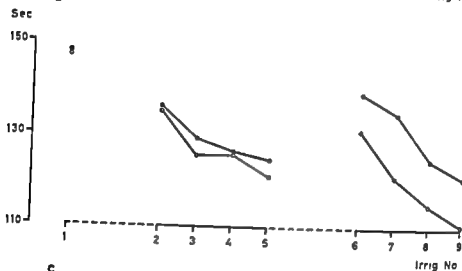
DISCUSSION

The decline of the vertigo and nystagmus responses to caloric stimuli reported at long intervals cannot be due to the influence of any one peripheral mechanism (e.g. sensory receptor adaptation, synaptic refractoriness or

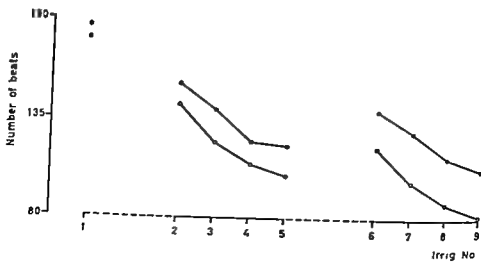
A



B



C



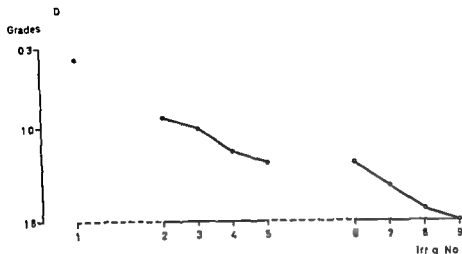


Fig 1 ■ Mean values for latency (a) duration (b) number of beats (c) and dysrhythmia (d) of nystagmus responses

○ = mean values for all observations ● = mean values after exclusion of series with Grade 3 dysrhythmia

fatigue) but indicates a central influence (increasing centrifugal inhibition or decreasing centrifugal facilitation)

The responses to repeated caloric stimulations showed some traits characteristic of habituation (1) Relative independence in respect of the magnitude of the interstimuli interval The initial strength of the response was not reached even after an interval of 15 days (2) Similarity to the learning curve (3) Specificity of response for different qualities of the stimulus (this was shown for the maximum intensity of vertigo in my previous series of experiments)

The following traits have been stated to be characteristic of habituation (1) Habituation is relatively independent of the magnitude of the interstimuli interval (Porter 1938 Harris 1943 Hood & Pfaltz 1954 Hernández Peón *et al* 1958 Hagbarth & Kugelberg 1959) This means that dishabituation is not strictly dependent upon the time factor in some cases dishabituation is still incomplete after the lapse of a considerable interval between stimuli (2) Habituation and learning curves are mainly alike (Dodge 1923 Coombs 1938 Lehner 1941 Harris 1943 Thorpe 1950 Hernández Peón *et al* 1957 1959) in that they are negatively accelerated (3) Habituation is specific for different qualities of the stimulus (Sharpless & Jasper 1956 Croen 1957 Hernández Peón *et al* 1957) but it may be generalized to some extent (Hilgard & Marquis 1940) (4) Habituation is influenced by centrally operative drugs (Tearing & Mowrer 1931 Hood & Pfaltz 1954 Hernández Peón 1959 Černý 1957 Hernández Peón *et al* 1957 1958 Zelenka 1960) (5) Habituation is influenced by certain psychological factors usually termed attention and excitement (Griffith 1920 Mowrer 1931 Prosser & Hunter 1936 Porter 1938 Wendt 1951 Hagbarth & Kugelberg 1958 Hernández Peón *et al* 1959 Palestini *et al* 1959 Larsson 1960) and may be influenced by hypnosis

(Platonov, 1930 Medvedevskij & Nevskij 1940—see Airapetyants & Kishyakov 1957) The close relation between habituation and conditioning has often been pointed out (e.g. by Mowrer 1934 Prosser & Hunter 1936 Lehner 1941 Hernández Peón *et al.* 1957, 1958 Hagbarth & Kugelberg 1958 Hernández Peón 1959)

It has been concluded from physiological and/or pharmacologic experimental results that *habituation depends on functions of the cortex* (Bechterew 1881 Asratjan 1940 Kolbe 1955—see Airapetyants & Kishyakov 1957 Smith 1941 Černý 1957 Zelenka 1960) *and of the brain stem* (King 1926 Sharpless & Jasper 1956 Palestini *et al.* 1959 Gernandt & Gilman 1960). A most differentiated opinion is expressed by Hernández Peón (1959). It is proposed that the brain stem reticular system plays a central role in that regulation. A feed back loop involving second-order sensory neurons which feed into reticular neurons and receive in turn centrifugal fibres from them may function as a reflex arc for sensory regulation. This circuit would receive higher modulating influences from the neocortex and the limbic system. Hagbarth (1960) points out that the reticular formation in its capacity as a diffuse activating system can hardly be held responsible for the habituation process which in a specific fashion affects the response to that particular stimulus which has been repeated. This phenomenon is probably more easily explained on the basis of specific feedback systems acting upon the sensory neurones. In respect of the vestibular system the findings of Engstrom & Versall (1958) and of Dohliman (1959) may be regarded as supporting this view to some extent. The former investigators found two distinctly different nerve terminals in the vestibular end organs and the later observed cholinesterase activity in the vestibular epithelium indicating the presence of cholinergic efferent nerve terminals.

A normally functioning vestibular system gives symmetrical responses to rotatory and linear accelerations and decelerations. It is well known that habituation to such natural stimuli occurs both physiologically (e.g. dancing skating movements of a ship on a rough sea) and experimentally (rotatory test). Under normal conditions the organism is probably exposed also to bilateral labyrinthine thermal stimulation from the surrounding air or water at least if there is a great difference in temperature between the organism and its environment. Hence synchronous irrigation of both ears with water at the same temperature should not be expected to evoke vertigo and nystagmus and as a matter of fact this does not usually occur (cf. Aschan 1955).

When the vestibular system functions *asymmetrically* (e.g. because of peripheral or central lesions or developmental anomalies) habituation can also occur (cf. Frenzel (1955) and Fluor (1960) on the effect of one-sided labyrinth destruction). From this standpoint the occurrence of habituation also to artificial mono labyrinthine thermal stimulations should not prove surprising.

ZUSAMMENFASSUNG

Versuche mit 50 jungen und aus otoneurologischem Gesichtspunkt normalen Individuen zeigten eine Abnahme der durchschnittlichen Vertigo und Nystagmus

Reaktionen auf wiederholte monoaurale kalorische Reizungen mit Wasser von 30°C

Diese Reaktionsabnahme traf sowohl mit kurzen (8 Min) als mit langen (24 Stunden oder mehr) Intervallen zwischen den Reizungen ein, was für eine zentrale Regulierung dieser Abnahme spricht. Bei statistischer Analyse erwiesen sich einige Züge, die als charakteristisch für die Gewöhnung (habituation) angesehen werden können.

Es ist von praktischem Interesse, dass die erste Reizung noch nach 2 Wochen eine signifikante Herabsetzung der folgenden Nystagmus-Reaktion bewirkte. Diese Herabsetzung zeigte sich deutlicher in der Latenz, Schlagzahl und Dysrhythmie als in der Dauer des Nystagmus.

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LYTIQUE COCKTAIL AND LOCAL ANAESTHESIA FOR BRONCHOGRAPHY IN INFANTS AND CHILDREN

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The authors use local anaesthesia combined with a lytique cocktail in bronchography of infants and children. The method and the experience obtained in 70 cases are described in detail and the advantages offered by the method are outlined. Notwithstanding the numerous advantages it is emphasized that the rigid criteria for establishing the indication for bronchography must be adhered to.

Bronchography (in the following BG) is gaining ever increasing acceptance in pediatric practice. One of the reasons why this is so is that the anatomical and many pathological changes not visualizable by means of the bronchoscope can be rendered visible by BG and thus can be evaluated better than by any other method of diagnosis. However, the use of BG is restricted by the technical difficulties involved. Most important of them is the elimination of the coughing reflex. Coughing interferes with the filling of the bronchi and the contrast medium may be sucked into areas and occlude bronchi that may severely impair respiration. Thus a good BG requires good anaesthesia which is difficult to achieve in early childhood because of the poor cooperation between doctor and patient. The coughing reflex of infants is weaker than is that of adults. For this reason no anaesthesia is used in infants for BG by some workers (1-3) while others (4-7) employ local anaesthesia even in infants.

However, we do not think this would achieve perfect anaesthesia for two reasons. One of them is that toxicity forces us to use lower doses and the other is the poor cooperation between patient and doctor. Earlier we too like LILL (8), Wishart (14), Love & Morow (9) had used ether anaesthesia when performing BG in infants and children. This method had the serious disadvantage that narcosis had to be very deep to eliminate the coughing reflex (narcosis had to be continued until the corneal reflex became extinguished) and the ether irritated the respiratory tract causing ample secretion and a sense of suffocation. Deep narcosis requires quick work and the contrast medium injected interferes with breathing causing an undesirable prolongation of narcosis after the completion of BG.

Since 1966 we have been performing bronchoscopy under deconnection. The method and the experience obtained have already been reported on

(11-12) In view of the good results we have tried out the method with BG as well. At first attempts were made to fill up the bronchi with the radiopaque medium under deconnection. This method proved to be inadequate because in the deconnected patient the coughing reflex is not extinguished and the contrast medium is usually coughed up even before the X-ray film could be made. In a few cases deconnection was combined with ether anaesthesia hoping that the parasympathetic effect of phenergan would counteract the hypersecretion caused by ether and also the dose of ether may be favourably reduced.

Unfortunately this type of anaesthesia resulted in apnea in several cases that could be relieved by artificial respiration and by the administration of excitants only. Finally we combined the laryngeal cocktail with local anaesthesia with excellent results. The procedure may be described as follows. The child is deconnected by the method employed with bronchoscopy i.e. of the cocktail laryngeal prescribed by Laborit & Huguenard (13) that contains 1 mg of larygectol, 1 mg phenergan and 0.2 mg dolantin or dolargan in each ml. 1 ml/kg body weight or 20 ml/square metre body surface area is injected intravenously. If the effect is not satisfactory one hour later half the above dose is given. If the response is satisfactory one third of the above dose is given for maintenance of deconnection at 20 minute intervals some times as often as two or three times. When the veins are in poor condition or are difficult to gain access to administration may be through the intramuscular route giving 1.5 times the above dose and repeating the doses at intervals of at least 30 minutes duration injecting on each occasion 1.5 times the intravenous dose. Intravenous administration may be combined with the intramuscular injection but in this case the first injection must be made into a vein and never into the muscle.

When deconnection has set in (the patient is asleep the muscles are hypotonic the tongue flutters the face is flushed and vasolability has developed) bronchoscopy is performed and the possibly present bronchial discharge is sucked off. Then through the bronchoscope tube about 1 or 2 ml of a 0.5% pantocaine solution is sprayed onto the bronchial mucosa the laryngeal catheter is inserted and the bronchoscope tube is removed. The patient is then transferred to the X-ray table and it is ascertained that the catheter is in the proper position. After turning the patient on one side further 1 or 2 ml doses of pantocaine are injected through the catheter into the bronchi. To remove pantocaine from the catheter air is inflated. After waiting 1 or 2 minutes the bronchial system may be filled up with the radiopaque medium. After having taken the pictures the elimination of the contrast medium is promoted by hanging the head and chest down and awakening is accelerated by the intramuscular injection of spiractin and caffeine.

A great advantage of the BG performed under deconnection is the relatively little risk involved. We think that the inhalation anaesthesia with ether and the intravenous anaesthesia with evipan succinyl etc. are much more dangerous. There are no unpleasant sensations (such as fear

example occurs with ether narcosis) and the total amnesia of the examination eliminates psychological alterations (such as fear) and the examinations may be repeated without version. Anaesthesia lasts $\frac{1}{2}$ to 1 hour without the administration of other narcotics thus the doctor has ample time and need not hurry. The antiemetic action of largactil rules out vomiting and defective swallowing during BG. Deconnection eliminates by reducing reflex hypersensitivity the potential danger of respiratory pressure and cardiac misregulation (shock, apnea). The decrease of bronchial secretion through the parasympatholytic action makes pre-treatment with sedatives or atropine unnecessary. The antihistamine effect (of phenergan in the first place) is thought to be of importance in the prevention of subglottic oedema.

Our experience obtained thus far may be summed up as follows. BG with lytiqne cocktail and local anaesthesia has been performed in 70 infants and children. None of the patients have been lost. BG could be performed undisturbed in every case and the bronchograms were good. If we did not wait until the pantocaine injected through the catheter took effect the contrast medium elicited coughing and as a result the bronchi were poorly filled and in some cases flew over to the opposite side. Deconnection does not eliminate every danger of BG and therefore in establishing the indication for its use the viewpoints outlined by one of the authors (12) must be taken into account. It is beyond doubt that deconnection must be performed by a specially trained paediatrist in an institute and even after completion of the BG examination careful medical and nursing control must be maintained for a few hours because of the danger of hypotension, apnea, intestinal paralysis and orthostatic collapse. In some cases BG may be followed by high fever, weakness and pallor and even pneumonia. This is due to the foreign body like behaviour of the contrast medium, the residues of which may cause retention of secretion and may promote the development of bronchitis, bronchiolitis and bronchopneumonia although propylidone is absorbed in 24 to 48 hours time. This explains why we try to accelerate the removal of the contrast medium after BG by turning the patient upside down and injecting coffeine and spiractin to cause the medium to flow out and to accelerate the return of the coughing reflex respectively. In view of the points made above it is absolutely justified to give antibiotics without delay in case BG is followed by fever.

ZUSAMMENFASSUNG

Bronchographien im Säuglings und Kindesalter werden von den Autoren in Localanaesthetie ausgeführt. Kombiniert mit Verabreichung eines Cocktail Lytiqne. Ihre Methode sowie die bei 70 Fällen gesammelten Erfahrungen werden ausführlich beschrieben und die Vorteile des Verfahrens erörtert. Ungeachtet der vielen Vorteile wird betont, die Bedingungen betr. Aufstellung von Indicationen zur Bronchographie strikt einzuhalten.

the experiments on the problem of etiology and treatment of some inner ear disease in relation to production of energy and shall present some discussions on them

The Role of Oxygen Deprivation or Vascular Disorder

In discussing the onset of inner ear disease the limiting factor in the maintenance of a stationary state of the energy production system in the inner ear should be taken into consideration. According to Koide (1) and Koide *et al* (2) it is considered that oxygen is the limiting factor and that oxygen deprivation in the inner ear caused by vascular accidents is probably the most important factor in bringing about some inner ear diseases.

Recently it was in fact revealed by Misrahy *et al* (3) and Koide *et al* (4) that oxygen tension in the inner ear of an animal decreases markedly during intense sound stimulation. If we combine the fact that sound stimulation decreases the oxygen tension in the inner ear with the reports by Tonndorf *et al* (5) and Beck & Beickert (6) that oxygen deprivation facilitates acoustic trauma it is acceptable that oxygen deprivation caused by sound stimulation may play an important role in the development of acoustic trauma.

The oxygen tension in the inner ear cannot be recorded quantitatively at present and its recording method has certain limitations. However according to Koide *et al* (4) the study of vascular disorder within the inner ear appears to be made possible when glucose in the perilymph is taken as an index because glucose content rises when an abnormal state is present in the vascular system of the inner ear.

Vestibular asymmetry

The long history of Meniere's disease began with the morphological findings in the inner ear suggestive of labyrinthine hydrops and the studies on this disease are very abundant. As to its etiology theories of neurovascular disturbances, allergy, focal infection, intoxication, imbalance of water and electrolyte metabolism and disturbances in vitamins and hormones have appeared and disappeared again and again. Since this disease is not fatal the patients live for a long time suffering from repeated attacks during which time secondary changes may complicate the picture. Accordingly it has been very difficult both clinically and experimentally to investigate the true etiology of this disease and many theories presented so far naturally do not go beyond mere assumptions.

As shown in Table 1 the duration (D) of postrotatory nystagmus (10 rotations/30 seconds) was measured in rabbits lightly anesthetized with urethane and accordingly the animals were divided into two groups, one in which the D values in the right and left rotations were equal and the other in which the D values in the right and left rotations were different. Just after rotatory stimulation the perilymph of both ears was collected by piercing the oval window membrane which was exposed directly with a dry glass capillary carried on a micro manipulator. The glucose contents were determined photometrically (4).

TABLE 1 Relationship between vestibular reaction and glucose content of perilymph

The glucose contents were determined by the method described in reference (4)

Group 1										
Rabbit no	III	87	91	97	96	101	83	90	99	98
Nystagmus ratio	1 0	1 0	1 0	1 0	1 0	1 0	1 1	1 1	1 2	1 3
Glucose										
mg%	R	138	113	147	148	200	103	182	179	129
	L	135	119	145	160	200	119	188	147	129
R/L x 100		107	95	101	93	100	86	97	88	100
Group 2										
Rabbit no	102	103	97	94	95	100	93	104	89	
Nystagmus ratio	1 5	1 5	1 6	1 6	1 6	1 7	2 0	2 0	2 0	
Glucose										
mg%	R	177	199	150	137	99	88	144	81	218
	L	119	125	157	144	6	79	138	79	206
R/L x 100		107	95	95	104	130	111	104	103	120
σ^2 population variance in mean value $F_2 \quad 3.82 \quad [F_2^0(0.05)] = 3.07 \quad (2.30 \quad t_2 = 2.20)$ $\sigma_1^2 < \sigma^2 \quad m_1 \quad m_2$										

As a result of this experiment it has been revealed that the animals with a small right left ratio of D represent a small population variance for a small right left ratio of glucose contents. From the viewpoint that the glucose levels are taken as criteria for determining normality or abnormality of the vascular system the animals with equilateral vestibular reaction appear to maintain the equilibrium of the vascular system in the bilateral inner ear.

This phenomenon will be further clarified by the results of the following experiments as shown in Table 2. By the statistical evaluation two cases of rabbits were found to be abnormal. In Case No. 89 the right left ratio of D was 3.1 and the glucose level in the perilymph on the right side was higher than the blood glucose level. Since the glucose level in the perilymph is usually lower than that in the blood the glucose level in the perilymph of the right ear is considered to be abnormal. In Case No. 224 the right left ratio of D was 4.2 and that of the glucose contents was 1.6 which are both in the abnormal range.

Vertigo is a subjective symptom and it is impossible to know if these animals experienced it. But the animals are assumed to feel the spontaneous vestibular vertigo which is induced mainly by the imbalance in the bilateral inner ear vascular system.

The balance of the glucose levels between the right and left inner ears is unstable and may be variable according to the general condition of the

TABLE 2 *Two cases of rabbits with imbalance of vestibular reaction*

(From Koide (1958))

Rabbit no	Nystagmus duration (sec)	Glucose (mg %)	
		Perilymph	Blood
89	Right rotation 7	Right ear 219	
	Left rotation 22	Left ear 206	
	Ratio 3:1		
221	Right rotation 9	Right ear 236	230
	Left rotation 38	Left ear 133	
	Ratio 4:2	Ratio 1:8	
Rejection limit (confidence coefficient 90 %)			
Duration 2.5 > ratio > 0.43			
Glucose 1.4 > ratio > 0.71			

TABLE 3 *Effect of anesthesia on glucose contents of the perilymph (Group B)*

Group A Rabbits anesthetized with urethane (these data are shown in Table 1) Group B Control (rabbits untreated)

Rabbit no	Glucose (mg%)		
	Right ear	Left ear	R/L × 100
222	128	148	87
223	88	109	81
224	226	133	181
225	89	100	89
226	75	64	117
229	114	100	114
230	119	130	92
231	138	130	106
232	103	110	93
233	91	111	85
234	122	130	91
235	130	136	96
239	97	78	124
240	100	85	118
$F_{11} = 4.58, [F_{11}^{10} (0.05) = 3.19]$ $\sigma_A^2 < \sigma_B^2$			

animal. As it seems probable that changes in the glucose content reflect the changes in the circulatory condition of the inner ear, the effect of constitutional conditions on the inner ear was examined by statistical analysis of the glucose levels of the perilymph as shown in Table 3.

The rabbits were divided into two groups: one group was anesthetized with urethane, and the other unanesthetized for control. Then the ratio of

glucose contents of the two ears was calculated in each animal. The result was as follows: the animals anesthetized with urethane, represented a small population variance for right-left ratios of glucose contents, and the control presented a large population variance. Therefore, when the reactions of the living body to endogenic or exogenic stimuli are blocked by treatment such as general anesthesia, the imbalance between the bilateral inner ear functions occurs less easily than when the animal is left untreated.

A consideration of the recurrence of Meniere's disease

Why recurrence of symptoms is so frequent in Meniere's disease is a hard question which has not been satisfactorily answered. From the viewpoint that the pathologic changes in the vascular system of the inner ear are the important factors of disease, the question is presented whether or not the susceptibility of the inner ear to abnormal conditions such as circulatory disturbances increases when the inner ear tissues are irreversibly damaged.

It is well known that streptomycin poisoning (7, 8), allergy (9) and hypoxia (6) produce tissue degeneration and increase the susceptibility of the inner ear to acoustic stimulation. As a result we might assume that inner ear diseases caused by vascular accidents or acoustic stimulation have a common etiological mechanism of disturbances in oxidative phosphorylation (4). Therefore it is reasonably considered that the impaired tissues are more easily damaged by vascular accidents and/or sound stimulation than the intact tissues.

Once Meniere's disease develops, even if vertigo disappears as a result of treatment, the biochemical changes in the inner ear may not recover and the susceptibility of the tissues to oxygen deprivation increases. In this situation the inner ear is in a susceptible condition to symptomatic attacks and slight circulatory disturbances which do not produce any damage in the normal ear may cause functional damage and a recurrence of symptoms.

In a normal ear oxygen deprivation causes a decrease in the oxygen tension in the inner ear, and when oxygen deprivation is removed, the oxygen tension recovers immediately to the initial level (4). However, in some cases the recovery did not occur or was incomplete, as shown in Fig. 1. Accordingly, the resistance of the inner ear to oxygen deprivation appears to decrease markedly when some damage is present in the inner ear. And it is considered that a slight degree of oxygen deprivation, which does not do any harm to the intact inner ear, may further aggravate the damage in cases of Meniere's disease.

A consideration of the Tullio phenomenon

The Tullio phenomenon, the appearance of vertigo on application of loud sound, is mainly observed in patients with Meniere's disease, although seen also in normal individuals. According to recent statistics of Nito (10), 5% of patients with Meniere's disease and 2.2% of normal individuals manifest this phenomenon. He attributed the cause to the decompression in the vesti-

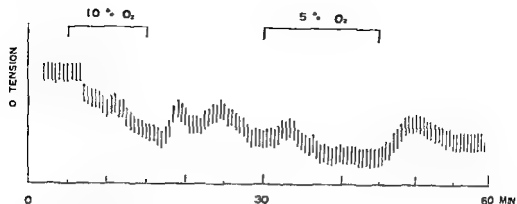


FIG 1 Abnormal effect of oxygen deprivation on the oxygen tension in the inner ear. A guinea pig weighing about 250 g was anesthetized with Nembutal. The platinum microelectrode was introduced into the scala tympani of the basal turn of cochlea and the oxygen tension was recorded.

bular system caused by sound pressure. In my opinion, decrease in the oxygen tension in the inner ear caused by loud sound is the direct factor in the development of this phenomenon. Then it is reasonably assumed that in cases of Meniere's disease the dysfunction of the inner ear, as in vertigo, may develop more easily by decrease in the oxygen tension in the inner ear than in normal individuals.

Thus it appears that there is a common etiological factor between the recurrence of Meniere's disease and the development of the Tullio phenomenon. In both cases, decrease in the oxygen tension in the inner ear is likely to be the direct factor. Although the fact that there is no Meniere's disease in animals makes studies of the cause of this disease very difficult, the results of my experiments are sure to be a clue to the understanding of Meniere's disease.

Biochemical Aspects of Treatment of Vertigo Deafness

On the basis of the experimental findings described so far, it seems reasonable to consider that oxygen deprivation in the inner ear caused by vascular accidents is one of the important factors in the onset of inner ear disease. Oxygen deprivation is probably the immediate determinant in disorders of inner ear function and may be regarded as the primary factor responsible for damage.

According to the results of our experiments, oxidative phosphorylation appears to be related closely to the metabolic activities of inner ear tissues. By the addition of both adenosine triphosphate (ATP) and diphosphopyridine nucleotide (DPN), the glucose oxidation was restored in the inner ear tissue in which it had ceased after a long period of anaerobic incubation. This shows that the restoration of oxidative phosphorylation is an effective measure for the recovery of tissue metabolism, and also suggests that oxidative phosphorylation is a limiting factor in the maintenance of tissue metabolism. This consideration was discussed in detail elsewhere (4).

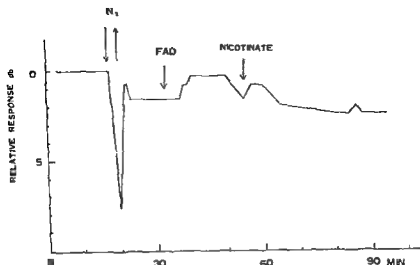


FIG. 2. Effect of vasodilator and cofactor on the depressed cochlear potentials caused by severe oxygen deprivation. A guinea pig weighing about 250 g was anesthetized with Nembutal. The recording microelectrode was introduced into the scala tympani of the basal turn of cochlea. The intraperitoneal dosages of agents administered to the animal were as follows: FAD 0.2 mg, nicotinic acid 1.5 mg.

The oxygen tension in the inner ear increases markedly by an injection of pilocarpin, nicotinic acid, ATP or sodium bicarbonate, and by stellate ganglion block. In general the metabolic cofactors did not appreciably improve the oxygen supply to the inner ear.

Observations were made on the depressed cochlear potentials of the impaired ears which had been produced by severe oxygen deprivation. As shown in Fig. 2 the injection of flavin-adenine dinucleotide (FAD) partially recovered the depressed potentials. And the potentials also recovered by the injection of nicotinic acid, but its recovery remained partial.

As a result it is suggested that after the removal of oxygen deprivation the oxygen tension in the inner ear does not always recover to the normal level, and that the depression of the cochlear potentials also may be due to the decrease in the metabolic activities of the inner ear even in the early stage of oxygen deprivation. In the case of acoustic trauma it was also observed that after the removal of sound stimulation the oxygen tension in the inner ear did not recover to the initial level immediately. Therefore it is essential that both circulatory efficiency and oxidative phosphorylation should be improved for the rational treatment of inner ear disease.

According to our clinical observation (11) it was concluded that the combined use of vasodilator and cofactor is the rational treatment of nerve deafness. However, because the earliest biochemical change in the damaged inner ear seems to be the inhibition of oxidative phosphorylation, it is expected that the combination of ATP and DPN will be the most successful of the

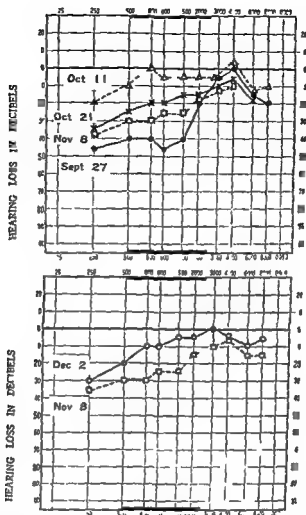


FIG. 3 Audiogram made on September 27 October 11 and 21 November 8 and December 2 in Case 1 with complaint of deafness in the right ear alone

therapeutic agents particularly in cases where the damages within the inner ear remains reversible

Recently a small amount of very pure DPN was offered to us by Kowa Chemical Laboratory Tokyo and this preparation was used as a therapeutic agent. It is well known that administration of excessively large amounts of ATP to animals causes lowering of blood pressure following a shock state. Our experiment has revealed that the 50% lethal dose of ATP is about 250 mg per 1g body weight (mouse). As a result of further experiment (12) it was revealed that DPN is practically non toxic although administration of extremely large amounts of DPN causes some histologic changes in the vascular system of the mouse and that addition of DPN to ATP does not practically influence the 50% lethal dose of ATP. This experiment made it possible to administer both ATP and DPN as the therapeutic agents.

The patients treated are the cases where deafness had not improved despite

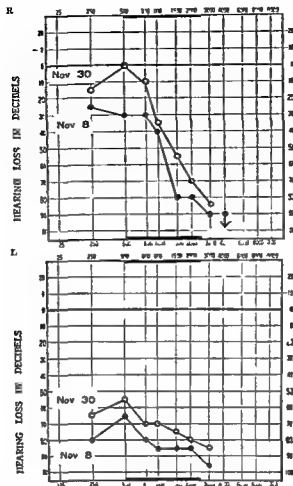


Fig. 4. Audiogram made on November 8 and 30 in Case 2 with bilateral nerve deafness

the administration of the usual therapeutic agents especially the vasodilator and cofactor. Twenty mg of ATP and 10 mg of DPN were dissolved in 40 ml of 20% glucose solution and injected intravenously every day. Eight patients were treated and three showed improvement of hearing. Representative cases are the following:

Case 1 (Fig. 3). Mr. M., aged 32, came to the clinic on September 27 because of attacks of vertigo and tinnitus associated with loss of hearing in the right ear. Symptoms had been present for four days. After 10 days of intravenous administration of lipoic acid solution and of stellate ganglion block the symptoms were relieved and on October 11 the audiogram revealed improvement in the right ear to ability to hear normal conversational tones. The treatment was continued but the patient complained again of progressive deafness on October 18. Because on October 21 the audiogram revealed loss of hearing again it was decided to give the intravenous use of both nicotinic

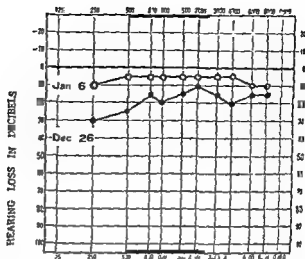


FIG 5 Audiogram made on December 26, 1960, and on January 6 1961, in Case 3

acid and lipoic acid another clinical trial. But this treatment was unsuccessful. Then the patient received first daily intravenous injections of both ATP and DPN from November 8 to 19, showing evidence of improvement and secondly intravenous injections of ATP alone from November 20 to December 11. As shown in Fig 3, the audiogram revealed definite improvement in the right ear.

CASE 2 (Fig 4) Mr. Y. N., aged 23, came to the clinic on January 1959 with complaints of deafness in both ears. The patient had noted deafness in both ears for about a year before he came to the clinic. With stellate ganglion block and intravenous injections of nicotinic acid, the hearing in both ears did not improve. He was unable to remain longer for treatment at that time but returned on November 1960 for further treatment. The patient received daily intravenous injections of both ATP and DPN from November 8 to 30. As shown in Fig 4, the audiogram revealed definite improvement in both ears.

CASE 3 (Fig 5) Mrs. C. Y., aged 51, came to the clinic on November 26, 1960, because of loss of hearing in the left ear. The appearance of the tympanic membrane was normal. Symptoms had been present for about 20 days. The patient was given both nicotinic acid and lipoic acid orally for two weeks. No change in symptoms and in audiogram occurred. Then she received daily intravenous injections of both ATP and DPN from November 15 to 26. An audiogram taken on January 11 1961, revealed definite improvement in the left ear.

COMMENT

Literature on inner ear diseases has swelled to the extent that even the most valuable paper cannot be introduced without omissions in such a short article as this. Therefore only a few reports that have been published

will be mentioned here. The theories mentioned by Proctor *et al* (13-14) that functional disorder of the inner ear can be brought about by autonomic imbalance or allergic conditions in the ear is used today as the basis of treatment of Meniere's disease, sudden deafness and nerve deafness.

According to Ruppmann (15) and Beickert (16) vasomotor dysfunction or sclerosis of blood vessels play an important part as underlying conditions of inner ear diseases and slow down labyrinthine circulation with resultant reversible or irreversible degeneration of the inner ear. Fowler (17) stated that the so called stress may cause severe neurovascular reactions resulting in local anoxia. It was pointed out by Williams *et al* (18) and Anderson & Rubin (19) that in the allergic reactions the effectiveness of the circulation in the shock tissues is impaired and there is a decreased delivery of oxygen to the capillary walls and the tissues.

In general these theories regarding the etiology of inner ear disease assume some vascular factors as a direct cause. In fact it was reported by Fowler (17-20), Williams *et al* (18) and Hallberg (21) that some vasodilators may produce relief in inner ear disease. However, no clear evidence was found to support the hypothesis that vascular disturbances are the important factor in bringing about inner ear disease and that vasodilators can produce an increase in the oxygen tension in the inner ear and recover the depressed cochlear function.

In the present investigation it has been revealed that the vascular phenomena which appear in the vascular system of the inner ear might play an important role in the onset of inner ear disease including acoustic trauma. Then in the early stage of inner ear disease biochemical lesions of the inner ear are classified as follows: (1) oxygen deprivation, (2) alteration of oxidative phosphorylation of the tissues.

Because successful management depends on the removal of these pathological circumstances if the changes remain in a reversible state the combined use of vasodilator and cofactor appears to be a rational method of treatment of inner ear disease.

The stimulation of lowered metabolic activities in the degenerated tissue may be achieved by the restoration of oxidative phosphorylation by the combined use of ATP and DPN. The effect of the combined use of ATP and DPN on nerve deafness suggests that the inhibition of oxidative phosphorylation is the limiting factor in degeneration of inner ear and that its restoration is the most effective measure for the restoration of hearing. I have stated that the degenerated inner ear tissue restores its respiration by the addition of ATP and DPN *in vitro*. But there are many problems regarding the role of ATP and DPN *in vivo* since the mechanisms of improvement of hearing following injections of ATP and DPN and the environmental factors responsible remain obscure in each patient.

Since the considerations described so far may be applicable only to the acute stage of disease the treatment of inner ear disease by the combined use of vasodilator and cofactor such as the combined use of ATP and DPN

naturally has certain limitations. Therefore, it is indicated that the problem concerning the chronic stage of disease must be approached from other angles.

Many problems are to be solved in the future in the field of biochemical studies on inner ear disease. Although the present study is only a very small part of the whole, the author has discussed the treatment of inner ear disease from the biochemical standpoint.

ZUSAMMENFASSUNG

Diese Untersuchungen haben gezeigt, daß vestibuläre Asymmetrie in engem Zusammenhang steht mit einem Missverhältnis hinsichtlich der Glukosebestandteile der Perilymphe zwischen dem beiderseitigen Ohr. Folglich wird angenommen, daß die Vestibularfunktion durch den Zustand der Gefäße im Innenohr beeinflusst wird. Dieses Ergebnis wird sicher zum Verständnis der Ménièreschen Krankheit beitragen, einschließlich Rückfall und Tulliosche Reaktion.

Da der Sauerstoffdruck im Innenohr auch während intensiver Schallreizung abnimmt, scheinen Störungen im Gefäßsystem und darauf folgender Sauerstoffmangel im Innenohr der wichtige ätiologische Faktor für den Beginn einiger Innenohrkrankheiten zu sein.

In diesen Fällen werden die biochemischen Beeinträchtigungen des Innenohres folgendermaßen klassifiziert: 1 Sauerstoffmangel, 2 Hemmung der oxidativen Phosphorylierung. Und es erwies sich experimentell und klinisch, daß die kombinierte Anwendung von ATP und DPN bei Innenohrkrankheiten von deutlicher Wirkung ist.

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GLOSSOPHARYNGEAL ZOSTER

SVEND HEIBERG

Ronne Denmark

A case of glossopharyngeal zoster in a 76 year old man is reported

A 76 year old artist consulted me at my office in Ronne Bornholm on January 4 1961, complaining of a rather severe left sided pharyngeal pain when swallowing the pain had been persisting for a couple of days. The patient had also been suffering from a headache and he had felt generally indisposed.

An examination of the pharynx disclosed on the palatine arch close to the left tonsil 5-6 whitish distinctly outlined ulcerations the size of hemp seed. An examination of the hypopharynx disclosed further 10 similar ulcerations also located on the left side and much resembling aphthae. The left side of the tongue base was coated by a thick, greyish yellow matter which I did not remove because of the soreness of the throat.

The patient was definitely in pain and his general condition rather affected. The left mandibular area was found to be the site of a swollen tender nodule but otherwise findings were normal.

The nasal cavity the larynx and the ears presented no pathological changes and the skin was normal without herpes.

Hitherto the patient had enjoyed good health and only once in 1959 had an operation ad modum Luc Caldwell been performed on account of a left sided dental empyema in the maxillary sinus which since then had been silent. To me the present throat symptom seemed rather peculiar and did not quite look like any ordinary aphthous stomatitis for which reason I made the preliminary diagnosis of virus infection of unknown nature. Hydrocortisone-succinate lozenges were prescribed 1 tablet qid.

One week later the patient was still complaining of a sore throat and also of severe stabbing left sided pains in his head. To me this symptom suggested the presence of a glossopharyngeal zoster.

On the following day (January 12) an examination disclosed that the lesions had healed completely around the left tonsil and to the left in the hypopharynx. The coating on the tongue had disappeared now making it possible to see several distinctly outlined partly coherent superficial but clean ulcerations distally to the left on the tongue and only to the left. New eruptions were not noted.

The patient described his headache as a stabbing burning exclusively left sided pain felt in the temple the forehead the tongue behind the left eye and radiating to the left ear.

The patient had felt rather dizzy mainly in the form of a general state of unsteadiness there were no signs of nystagmus and the caloric reaction was normal. Negative Romberg's symptom. The sense of hearing was unaffected.

The gustatory sense was peculiarly disturbed the patient could not eat anything salt and said it burned in the throat like hydrochloric acid. Best of all was sweetened food, sour food tasted as usual, bitter food had not been tried.

For some days the evening temperature had been above 38°C . Eight days later the tongue ulcerations had healed completely and new eruptions had not occurred. According to the patient the sensitivity of the throat was uniform in both sides. The gustatory sensations and the neuralgic pains persisted.

Fourteen days later i. e. more than one month after the onset of the disease the gustatory sensations had subsided and the pains were no longer troublesome. Now the most essential complaints included fatigue and nausea, two symptoms which had been manifest throughout the period of disease. The patient, who is an artist of note and still at the height of his career, had taken up painting again. No eruptions of zoster and no occurrence of varicella had been diagnosed in the patient's environment.

It can hardly be doubted that this case was a glossopharyngeal zoster. The four symptoms required for this diagnosis are: (1) the occurrence of herpes zoster is unilateral; (2) the eruption occurs then and there, not by stages; (3) the location is the innervation area of one or more nerves; and (4) neuralgic pains in the same area are manifest. All of these four symptoms were noted in the present case.

A review of the otological publications from the past decade discussing cases of cranial nerve zoster shows that several cases of otic zoster are on record, also a few cases are described of zoster eruptions on the frontal two thirds of the tongue with or without accompanying facial palsy. Jepsen, for example, reported a case to the Danish otological Society in 1909 (1) but I have never seen any reports of the glossopharyngeal zoster (z. gl.) nor have I seen any descriptions of this feature in the several medical and neurologic text books.

Indeed, it would be unreasonable to imagine that the disease had never before been seen. de Jong states in his circumstantial text book on neurology (2) under the heading of pathology of the glossopharyngeal nerve that herpes zoster in the petrous ganglion has been seen but represents a rare finding. The sources of de Jong's knowledge remain unknown to me. According to this author the neuralgia represents the most essential disorder of the glossopharyngeal nerve.

This might suggest that occurrences of glossopharyngeal zoster are not quite as rare as hitherto presumed, they may have been neglected and hence some cases of neuralgia may actually have been cases of herpes zoster. A routine examination of the throat will disclose the eruptions around the tonsils, a symptom which easily may be confused with aphthous stomatitis.

Lesions on the distal third of the tongue are less easily observed and eruptions in the hypopharynx can only be diagnosed by speculum.

The greatest advantage of making the proper diagnosis is that it will provide an explanation of the rather severe neuralgias and thus the patient will be spared a series of unnecessary examinations and the physician will be saved his diagnostic troubles.

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STUDIES ON THE SENSE OF SMELL AND TASTE IN DIABETICS

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Qualitative and quantitative studies on the sense of taste and smell in diabetic patients did not reveal any changes in the sense of taste. The sense of smell was studied in 58 patients and the results were compared with a normal series. Thirty-five (60%) showed a definitely impaired sense of smell including 24 with severe impairment. Seven had anosmia. There was no correlation between the hyposmia and the patients' ages or between the hyposmia and the duration or severity of the diabetes. At present, the cause of the impaired sense of smell is unknown.

That diabetics may have disturbances of the sense of smell and taste has been known for a hundred years. Jordao (1857) found impairment of smell and taste as an initial phase of diabetic coma in a patient who was also suffering from impairment of vision and hearing. Among 11 diabetics Kulz (1874) found one suffering from a disturbance of taste. In 1899 the same author in his monograph comprising the case histories of 692 diabetics reported that a large proportion were suffering from impaired sense of smell and taste and a few complained of abnormal sensations of smell and taste. In Kulz' opinion this was due to changes of the nasal mucosa as well as of the peripheral and central nervous apparatus. Senator (1876) believed that apart from changes of the nasal mucosa and nervous apparatus an altered state of the oral secretions might be responsible for changes in the sense of taste in diabetics. von Noorden (1912) found changes of the sense of smell and taste to be uncommon and to play a subordinate role in diabetics. In six of his own patients he observed a complete loss of the sense of taste introductory to fatal diabetic coma. In rare cases he had observed paraesthesia. For instance, two of his patients were annoyed by a taste of bad eggs.

In these papers no mention is made of the technique of investigating the functions of smell and taste. Often the statements are probably based only upon the patients' subjective sensations. A taste of bad eggs is probably interpretable as a disturbance of smell.

The first quantitative studies on the sense of smell and taste in diabetics are from the last 10 years. Vigi in 1930 studying the sense of smell in 150 diabetics by the Isberg & Levy olfactometer found changes in 80%. Out of these 80% 33% had a decreased and 47% an increased sense of smell.

Such changes were commoner in older than younger patients. Ancona (1950) also found changes of the sense of smell in 80 % of his patients. Three quarters had hyposmia and one quarter hyperosmia. The hyposmia he interpreted as being due to lesions of the olfactory nerve and centres whereas the hyperosmia was stated to be due to increased irritability in the peripheral receptors due to inflammatory changes of the nasal mucosa. Vidau (1950) examined 70 diabetics also by the Elsberg method, all of whom proved to be suffering from mild hyposmia unrelated to the severity of diabetes. According to this author, it was due either to olfactory neuritis or congestion of the nasal mucosa.

The sense of taste was studied by Bonatti (1950) in 150 diabetics by a semiquantitative method using various concentrations of bitter, salt, acid and sweet gustatory stimulants. Fifty-four had a normal sense of taste for all qualities while 96 gave abnormal reactions. There was no relationship between an altered sense of taste on the one hand and the patients' ages or the severity of their diabetes on the other. More patients had increased than reduced sense of taste. The abnormal findings were put down to peripheral or central degenerative nerve lesions.

In a previous paper we have reported the results of a study on the inner ear function and cranial nerves in 69 diabetic patients. An oto-neurological investigation must also include testing of the sense of smell and taste qualitatively as well as quantitatively. Below, the results will be reported. Out of the 69 patients 23 were referred to us from the Steno Memorial Hospital and 26 from the Anterior Clinic of the University Hospital which accounts for the female preponderance. The material also is not representative of the age distribution of diabetics since we preferred including young patients with a long history of diabetes in order to avoid as far as possible interference by the physiological changes of aging.

For details regarding the past history and objective findings in respect to the diabetes to which we have attached great importance in the course of our studies, reference must be made to the previous paper. In addition the patients were questioned about subjective alterations in the sense of taste or smell and about present or past diseases of the nose and nasal sinuses. Anterior and posterior rhinoscopy, pharyngoscopy and laryngoscopy were performed in all cases. The sense of taste was investigated qualitatively for sweet, acid, salt and bitter by saccharose, sodium chloride, citric acid and quinine hydrochloride respectively. Quantitatively the sense of taste on the anterior part of the tongue was determined by the electrogustometer advocated by Krarup (1949).

The sense of smell was studied on the lines advocated by Zilstorff-Pedersen (1947). In the qualitative study we made the patients identify known odorous substances, both those which convey pure impressions of smell and substances which exert a trigeminal stimulation effect. For the quantitative study we used the apparatus of Elsberg & Levy (1935). As an odorous substance we used coffee which is a pure olfactory stimulant.

RESULTS

The table gives the results of the studies on the sense of taste and smell in our series shown in relation to the patients' ages, the duration and treatment of diabetes, and the late complications in terms of the degree of retinopathy. As already mentioned, the patients are the same and listed in the same order and by the same numbers as in our previous paper on the inner ear functions.

A normal qualitative sense of taste is indicated by —, lost sense of taste by +. The threshold values for the quantitative measurement on the right and left side of the tongue are stated in electric gust units (cf. Krarup 1958).

A normal qualitative sense of smell is indicated by —, and a lost sense of smell by +. The threshold values for the right and left side of the nose which indicate minimum perceptible odour are stated in ml. For graduation of hyposmia, see below.

Out of our 69 patients only 62 had the gustatory studies. For the sake of perspicuity, all the patients are listed in the table.

The qualitative gustatory test revealed a normal sense of taste in all but three patients who had lost the sense of taste for all qualities. All three were over 60 years of age. Electro-gustometric study disclosed a marked dispersion of the threshold values within each age group. Comparison with Krarup's (1958) normal series showed no difference. Thus, when investigated by the methods used, there is no difference between the sense of taste in diabetics and normal persons. A significant difference between the two sides, i.e. a difference in the threshold values on the right and left side of the tongue of more than 3 electric gust units, was found in only one patient, Case 66, in whom this difference was 6 electric gust units. The qualitative gustatory test showed loss of the sense of taste, so that the finding cannot be attributed with any value.

The sense of smell was investigated in 61 out of the 69 patients. In three of the cases, rhinoscopy showed the nasal mucosa to be so altered that no importance can be attached to the results of the olfactory test, and these results are given in brackets. The mucosal changes in these three patients were due to acute rhinitis, chronic maxillary sinusitis, and chronic hypertrophic rhinitis. In all the remaining patients, the nasal mucosa was of normal appearance. Of the remaining 58, seven were found to have lost their sense of smell. In the quantitative studies of smell, the threshold values were compared in each case with the values found for the corresponding age group in Kristensen & Zilstorff-Pedersen's (1953) normal series. Values on the verge of pathological were considered normal. Definite impairment of olfactory sense is indicated by + in the table, severe impairment by ++. It will be seen that 23 patients had a normal sense of smell and 35 (60%) a definitely impaired sense of smell. Twenty-four out of these 35 patients even showed a greatly reduced olfactory sense, and seven total anosmia. In all cases, the loss was bilateral, and there were no significant differences

TABLE 1 *Olfactory and gustatory examinations in diabetic patients*

Case no	Age of patient	Duration of diabetes	Duration of insulin treatment	Retinopathy	Gustatory sense (qualitative)	Gustatory sense in electrogustometric units r/l	Olfactory sense (qualitative)	Olfactory sense quantitatively in ml for coffee r/l	Graduation of hypoxemia	Anaesthesia
1	16	7	7	+	-	10/12	-	5/5	0	
2	16	5	5	0	-	10/12	-	12/13	+	
3	17	12	12	0	-	7/7	-	20/20	++	
4	17	12	12	0	-	7/7	-	20/20	++	
5	17	9	9	0	?	?	+	?	?	
6	19	9	9	0	-	7/7	-	?	?	
7	19	11	11	+	-	7/7	-	10/10	0	
8	20	6	6	++	-	11/14	-	>30/>30	++	
9	20	14	14	0	-	7/7	-	30/30	++	
10	21	4	4	0	-	7/7	-	15/15	+	
11	22	15	15	+	-	7/7	-	8/8	0	
12	22	16	16	++	-	27/27	-	5/5	0	
13	23	15	15	++	-	7/7	-	10/10	0	
14	23	16	16	+	-	3/3	-	5/5	0	
15	23	8	8	0	-	7/7	-	30/30	++	
16	23	17	17	++	-	7/7	-	5/5	0	
17	24	4	4	(+)	-	10/7	-	25/20	++	
18	25	13	13	+++	-	7/7	-	3/3	0	
19	25	9	9	++	-	7/7	-	20/20	++	
20	26	11	11	++	-	7/7	-	30/30	++	
21	26	15	15	++	-	7/7	-	20/15	+	
22	27	1	1	0	-	7/7	-	>30/30	++	
23	27	14	14	0	-	7/7	-	8/8	0	
24	27	3	3	0	-	21/21	-	?	?	
25	27	5	5	0	-	3/3	-	3/3	0	
26	28	0	0	0	-	3/3	-	>30/>30		
27	28	26	26	0	-	21/23	-	(>30/>30)	(+)	(+)
28	28	4	4	0	-	7/7	-	20/25		
29	28	14	14	0	-	7/7	-	30/30		
30	28	13	13	+++	-	5/7	-	5/10	0	
31	30	15	15	0	-	7/7	-	5/5	0	
32	30	22	22	++	-	7/7	-	30/30		
33	31	15	13	++	?	?	?	?	?	
34	31	3	3	0	-	7/7	-	10/10	0	
35	31	19	19	+	-	?	?	15/30		
36	31	27	27		-	7/10	-	5/7	0	
37	34	18	18	-	-	10/7	-	15/20		
38	34	32	32	++	-	7/7	-	10/10	0	
39	34	1	1	0	-	7/13	-	>30/30		
40	36	18	18	0	-	8/10	-	20/10		
41	36	21	21	-	-	?	?	?		

TABLE 1 (continued)

Cas. no	Age of patient	Duration of diabetes	Duration of insulin treatment	Retinopathy	Gustatory sense (qualitative)	Gustatory sense in electrogoniometric units r/l	Olfactory sense (qualitative)	Olfactory sense quantitative in ml for coffee r/l	Graduation of hyposmia	Anosmia
42	36	30	30	+	-	7/12	+	>30/>30	++	+
43	39	2	2	0	-	7/4	-	10/20	+	
44	39	30	30	++	-	17/17	-	8/10	0	
45	40	4	4	+	-	4/4	+	5/5	0	
46	41	29	29	++	-	16/18	+	(>30/>30)	(++)	(+)
47	41	4	4	0	-	10/10	-	20/20	+	
48	44	14	14	+	-	5/5	-	7/7	II	
49	45	22	22	+++	-	7/7	-	>30/>30	++	
50	47	13	13	++	-	10/7	-	30/>30	++	
51	48	12	12	0	?	?	?	?	?	
52	48	29	28	+	-	24/21	-	10/10	II	
53	49	33	33	+	-	7/7	-	20/20	+	
54	50	21	21	+++	-	10/10	+	>30/>30	++	+
55	51	6	6	+	-	25/25	+	(>30/>30)	(++)	(+)
56	52	21	21	++	-	11/15	-	20/20	+	
57	53	4	4	0	-	19/22	+	>30/>30	++	+
58	58	8	8	0	-	17/17	-	10/20	0	
59	59	II	8	0	?	?	?	?	?	
60	59	33	31	++	-	24/24	-	15/20	+	
61	59	3	3	++	-	34/36	+	>30/>30	++	+
62	59	29	29	++	-	10/10	-	15/15	+	
63	60	20	15	++	?	?	?	?	?	
64	60	6	0	0	-	7/11	-	5/10	II	
65	61	1VI	0	0	+	37/37	+	30/33	++	+
66	69	5	0	0	+	24/18	+	30/30	++	+
67	70	5	0	0	+	37/37	+	>30/>30	++	+
68	70	8	0	0	-	20/12	-	>30/>30	0	
69	73	13	13	+++	-	20/20	-	20/20	II	

between right and left. Out of 39 patients under 40 years of age, 22 (58%) had hyposmia which was present in 13 out of 20 patients over 40 (65%). In other words, there is no definite correlation between the patients' ages and the hyposmia after correcting for the age conditioned physiological impairment of the sense of smell. There was no correlation between the duration of diabetes and hyposmia and also no correlation between hyposmia and the late complications of diabetes in terms of the degree of retinopathy.

COMMENTS

While our investigations of the sense of taste in diabetic patients did not reveal any deviations from normal, we found that frequently diabetics show an even severe impairment of olfactory sense.

Although a few of our patients stated that their sense of smell was impaired the great majority were not aware of their hyposmia. None of them complained of parosmia. This is perhaps the explanation of the paucity of studies on the sense of smell in diabetics.

The reason why, unlike other authors, we did not find a relation between hyposmia and age, we believe, is that by a comparison with a normal series we corrected our results for the age conditioned physiological decrease in the olfactory sense.

It is difficult to give a reason for this impairment of the sense of smell. Neither we nor others have found any relationship between the duration of diabetes and hyposmia, and also no correlation has been found between the hyposmia and the severity of diabetes or its late complications. The series are small, however, and possibly such a relationship might be shown by larger series.

Impairment of the sense of smell like hearing impairment may be of the conduction type, caused by changes of the nasal mucosa or of the perception type in which case it would be caused by changes in the peripheral receptor cells or in the central nervous pathways. In our series rhinoscopy did not reveal conditions differing from normal. This of course does not rule out that histochemical and/or histological studies of the nasal mucosa might find the explanation at this site. To our knowledge no such studies have been reported, and also no studies of the central olfactory tracts in diabetics. It would seem rational to look for vascular changes chiefly in the capillary network similar to the changes which have been found to such a great extent in other diabetic complications. Pending such investigations the discussion on the cause of the impaired sense of smell in diabetics must be purely hypothetical.

ZUSAMMENFASSUNG

Änderungen des Geschmacksinnes bei Diabetikern konnten weder durch qualitative noch durch quantitative Untersuchungen erhoben werden. Der Geruchssinn wurde an 58 Patienten untersucht und die Resultate mit Normalen verglichen. 30 (60%) Patienten hatten einen sicheren verringerten Geruchssinn und von diesen konnte an 24 Patienten ein stark verringerten Geruchssinn festgestellt werden und an 7 Anosmie. Es konnten keine Relationen zwischen Hyposmie und Alter des Patienten oder zwischen Hyposmie und Länge und Schwerheitsgrad des Diabetes festgestellt werden. Bis heute weiss man nichts über die Ursachen des herabgesetzten Geruchsinnes.

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PROFUSE EPISTAXIS IN HYPERNEPHROMA METASTASIS

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It is pointed out that reports of hypernephroma metastases in the nose and paranasal sinuses are rare. On the basis of four personal cases it is suggested that such metastases are, however, presumably far more common in this region than can be inferred from the literature.

An account is given of certain characteristic properties of both hypernephroma and its metastases that hamper an early diagnosis. It is stressed that treatment of these metastases in the nose and paranasal sinuses presents special problems in view of the great tendency to haemorrhage.

Most cases of epistaxis present neither diagnostic nor therapeutic problems. Once the bleeding vessel has been localized the bleeding can usually be arrested without difficulty. In certain cases, however, the aetiology is uncertain and further investigation is required. The cause may be a tumour—either benign or malignant—and roentgenologic examination of the paranasal sinuses is therefore made routinely.

Although epistaxis is generally stated to be an early sign in carcinoma of the nose and paranasal sinuses this does not in fact seem to be the case. It is true that epistaxis occurred in about one third of all cases in the series reported by Larsson & Mörtensson (1954) but it was usually a late manifestation. Epistaxis as an initial symptom was present in only about 8 per cent of the cases. Despite this, nose bleeding must be regarded as an extremely important sign in carcinoma of the nose and paranasal sinuses.

Metastases from distant tumours are rare in this region but cases have been described in the literature of metastases from e.g. mammary carcinoma (Ungerecht 1950), carcinoma of the stomach (Hommerich 1954), seminoma (Garrett 1960) and hypernephroma (Bjendira 1951 among others).

Hypernephroma metastases in particular are characterized by a marked tendency to bleeding so that even cautious biopsy may give rise to life threatening haemorrhage.

Since the primary tumour may lie latent for many years it often remains undetected until symptoms from metastases make their appearance (Riches *et al.* 1951, Lundgren 1960 among others). Thus if the metastatic growth is localized to the nose and paranasal sinuses epistaxis may be the symptom leading to diagnosis of the primary malignant tumour of the kidney (Bjendira 1951, Burns *et al.* 1956, Garrett 1960).

In study literature, we find two types of responses to the situation of the male and parental union. One of them was treated at the Faculty of Natural Department, Karlsruhe (Germany) and was written by Hammer (1949). De Lury (1950) gave an account of it after Garbutt's earlier, and also a personal case in a letter Hammer (1950). Both H. G. Bours et al. (1950) and Garbutt (1950) have each reported one case.

The first is that the Commission has not yet received the necessary information from the various countries to enable it to make a final decision on the matter. This is probably because the Commission has not yet received the necessary information from the various countries to enable it to make a final decision on the matter.

Case Study

Case 1

Since this case has been covered in detail by Washington Post, only a brief account will be given here. The patient was a 35-year-old woman, who was married to the Earl, wife of the State Department. Her husband, a member of Congress, was a member of the Senate. Several specimens had been taken from the body of the patient in the early stages of the disease, but only one, which was a small, white, irregularly shaped, firm, nodular mass, was found to be a carcinoma. The patient died of the disease on June 1, 1941.

The patient has been covered in a warm blanket and a heating pad to the feet. The examination of the lungs and heart is normal. The patient is alert and oriented. The patient is comfortable and has no pain. The patient is being monitored for any changes in vital signs and level of consciousness.

Group 2

[illegible]

The patient was referred to hospital and admitted on February 1, 1964, with the following complaints: "I have been having a lot of trouble with my stomach and I have been losing weight. I have been having a lot of trouble with my stomach and I have been losing weight. I have been having a lot of trouble with my stomach and I have been losing weight."

[illegible]

Even though no primary tumor had been demonstrated, a diagnosis of metastasis was already suggested at autopsy. The specimen was then used for histologic examination and showed the presence of a malignant tumor.

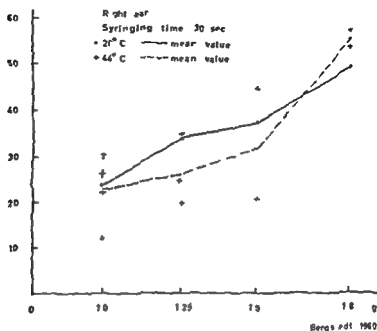
Since the patient's general condition was not improved by the above therapy, the patient had no further therapy.

Caloric tests in human centrifuge

Maximum intensity
of nystagmus

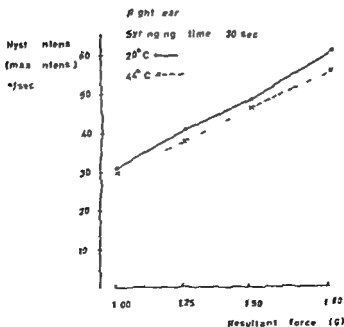
Four test subjects

°/sec



Caloric test in human centrifuge

G B



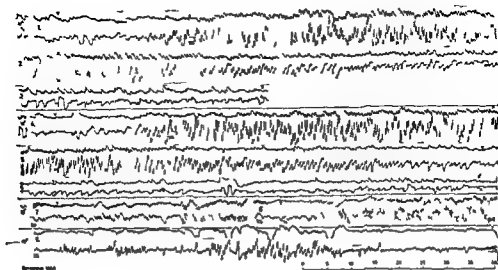


FIG. 5. Electronystagmographic recordings during caloric tests in the human centrifuge from three subjects. All records were performed with right ears. The records read from left to right: *V*—recording of vertical eye movements; eye movement upwards is represented upwards in the trace; eye movement downwards is likewise downwards in the trace. *H*—recording of horizontal eye movements; eye movement to the right is upwards in the trace; eye movement to the left is downwards in the trace. *S*—irrigation; *SS*—irrigation (syringing) stops. In the left margin is calibration for 10° eye movements in the vertical and horizontal traces. In the lower corner right is time in seconds (paper speed is constant at 1 cm/sec).

One caloric test with cold water (20°) at 1.50 *g* in the continuous record marked in the left margin with 1, 2 and 3. In horizontal trace nystagmus to the left (rapid phase downwards).

Similar test at 1.80 *g* with the same subject in records 4, 5 and 6. Note the higher maximum intensity of nystagmus at the higher *g*-bracket.

Records 7 and 8: another subject and warm water at 1.0 *g* (record 7) and 1.80 *g* (record 8). In picture are shown only parts of the records after irrigation (*SS*). Note the much higher maximum intensity about 35 sec after *SS* in record 8 (1.80 *g*) than 35 sec after *SS* in record 7 (1.0 *g*). This subject shows some tendency to dysrhythmia.

Even more marked dysrhythmia is seen in record 9 with the third subject shown in this figure. Cold water. This was the subject who had undergone earlier centrifuge tests.

produced in caloric tests with cases of dysrhythmia described by Aschan, Bergstedt & Stihle (1956). The inhibiting tendency was more pronounced with higher *g* loads. The latency time was noted. There was a certain interference during some recordings which rendered them unreliable; however, those which are reliable are given diagrammatically in Fig. 6. With higher *g* load the latency is shorter.

The duration of nystagmus shows a clear tendency to increase with higher *g* loads. With high *g* loads (1.50–1.80 *g*) it exceeds the 3 minutes during

FIG. 3. Maximum intensity of nystagmus in caloric tests in relation to *g*-value in all the 27 caloric tests made with four test subjects. Unbroken line is mean value of tests with cold water (20°) and broken line with warm water (41°).

FIG. 4. Maximum intensity of nystagmus in caloric tests in relation to *g* value in one and the same subject. Markings as in Fig. 3 above.

Latency of nystagmus in caloric tests in human centrifuge

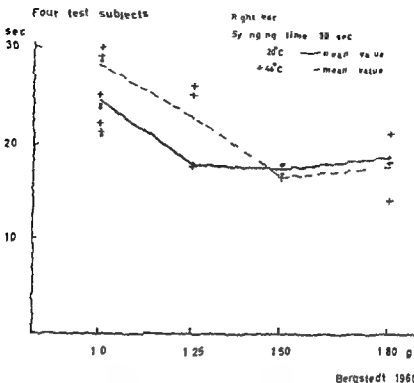


FIG. 6. Latency of nystagmus in caloric tests at different g brackets. Unbroken line is mean value from cold water tests and broken line from warm water tests. Latency is shorter at higher g .

which recording was carried out and this fact has unfortunately made it impossible to give a mean value for the increase in nystagmus duration.

Subjective sensations during the caloric test were described as a weak sensation either of tipping or of wheel rotation with cold water irrigation towards the right and with warm towards the left. Both types of sensation were markedly stronger the higher the g that was being pulled. It was not possible to form a clear comparison as to whether the tipping sensation increased more noticeably than the rotating one. During the caloric test there was in no case a sensation of rotation round the axis of the centrifuge nor of linear acceleration.

DISCUSSION

The method used in this experimental series to increase the strength of the gravitational field by means of a large centrifuge has been in use for many years in physiological studies. It has also been used to a lesser extent in vestibular research in which the sensations and oculogyril illusion were studied in conjunction with high accelerations and large g loads (Gravlund & Hupp 1946; Brandt 1957).

In research into positional nystagmus the present author (Bergstedt 1957, 1960a and b, 1961) has shown that as a result of a momentary increase in the strength of gravitational force in a human centrifuge there is an increase

in the intensity of positional nystagmus and that this increase stands largely in proportion to the relative increase in the value of g . He further showed that the only way to achieve a change in positional nystagmus intensity was to produce a change of g and that subjects without positional nystagmus before the centrifuge test showed no nystagmus in the centrifuge. His interpretation of this was that a change in the stimulation intensity of the otolith organ was the deciding factor in the intensity of positional nystagmus. In the present experiments we are concerned with caloric stimulation of the semicircular canals carried out at various g loadings in which case the maximum intensities of the nystagmus stand in direct proportion to the numerical g load. This points to different receptor organs stimulated in the two types of experiments.

A theory has been put forward to the effect that caloric test nystagmus is the result of the cooling or warming of the receptors in the otolith organ (Borries 1925). But on these lines it seems very difficult to explain the reversibility of the reaction when warm or cold water is used at various g loads. In the positional nystagmus experiments mentioned it was shown that stimulation of the otolith organ produced nystagmus in proportion to the relative g loading while with stimulation of the semicircular canals it was in proportion to the absolute g load. On the other hand that the caloric test may have influence on the otolith organ seems plausible especially when one considers the sensations of tipping that some test subjects experience (Jongkees 1948). In the present experiments the weak sensation of tipping was more pronounced with higher g values. The nystagmus following calorisation can hardly be explained by otolith stimulation however. If influence of temperature changes on the receptor cells could occur in the receptor cells of the otolith organs it would also be reasonable to expect a similar sensitivity to temperature in the cells of the cupula organ and that warm water would lead to a more marked nystagmus than cold. However according to Stahle (1958) cold and warm water produce equally marked nystagmus intensities. It seems difficult to conclude that the sensation of tipping depends on otolith stimulation and nystagmus on cupula stimulation.

The present experiments were carried out at water temperatures of 20° and 44°C. In order to arrive at an answer to the question posed at the outset of the research it was not considered necessary to use the temperatures of 10° and 44°C which are used in Hallpike irrigation. The temperature of the water used in the caloric test is of the greatest importance as far as the resulting nystagmus intensity goes (Stahle 1956 Jongkees 1953 Hamersma 1957). In order to achieve the most exact temperature regulation possible with cold water the room temperature of the centrifuge cabin was chosen. But when it came to warm water the temperature was thermostatically regulated not however to within the same limits as was possible with cold water. The results of the investigation though lead to the conclusion that the nystagmus is of equal intensity when plus or minus differences between water and body temperatures are equally large.

The caloric test carried out in a centrifuge by Hallpike's method should however be able to be used in investigating the causes of preponderance and the writer is at the moment engaged in an investigation of clinical cases with preponderance. The present author has found (Bergstedt 1961) in centrifuge experiments that preponderance in many clinical cases increases with higher g loads and has concluded that the otolith organ is in many but not all cases contributory to preponderance. Hallpike (1942) has earlier put forward a similar hypothesis to the effect that preponderance can be traced to the otolith organs or to an asymmetric function of them (utricle paresis).

In order to avoid Coriolis acceleration the subject's head was kept quite still. At high g loads the increased body weight made this easier and the increase in giddiness and tipping sensations which the subjects felt led therefore to no difficulties. A closed circuit television camera placed in front of the subject in the cabin—the screen being at the experimenter's side—made checking of the head position easy. The sensations cannot therefore be explained as being due to the influence of Coriolis acceleration arising out of movements of the head. Besides such acceleration has a tangential direction and can be compared with tangentially aligned acceleration or retardation (Bergstedt 1961). As is shown in Fig. 2 the horizontal plane of the semicircular canal lies at a right angle to the tangent and thus no influence on the currents in the horizontal canal can be expected to arise.

According to Bárány the direction of endolymph currents is reversed according to whether warm or cold water is used, providing the position of the head is not altered. Seen from the centre of the centrifuge this means that the endolymph currents will have opposite directions. However the experiments show that with caloric tests carried out in a centrifuge the intensity of nystagmus increases by the same amount with higher g loads whether warm or cold water is used. And though in this case the warm and cold temperatures were not distributed evenly on either side of the body temperature Fig. 3 points to this conclusion.

According to Quix (1929) the influence of the temperature differential on the vertical semicircular canal gives rise to the rotational component of nystagmus during a caloric test. However the method used in the present research does not record purely rotational components (Asch in Bergstedt & Ståhle 1950 and others). Yet a possible vertical nystagmus movement as a result of stimulation to the vertical canal is not unthinkable. In these experiments recording of vertical movements was also carried out. Such vertical movements are apparent with the normal caloric test when carried out on the experimental couch with irrigation of the right ear with cold water the nystagmus often occurs upwards and to the left with warm water downwards and to the right. The present experiments (Fig. 5) suggests that with higher g loads the nystagmus intensity is increased by a proportional amount in both the horizontal and vertical directions.

In the present case the best results were achieved with cold water syringing

Maximum intensity
of nystagmus

Caloric tests in human centrifuge

Four test subjects

Light on

Spring time 30 sec Cold water 20°C

— Mean values

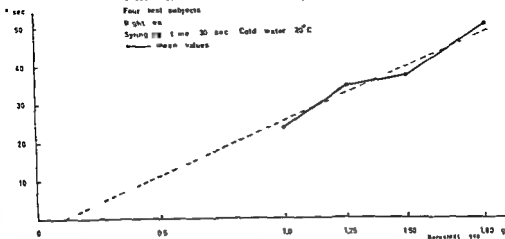


FIG. 7. Mean values of maximum intensity of nystagmus in caloric tests with cold water (20°C) in relation to g -value (the same as in Fig. 3). Broken line marking an extrapolation to g -values below 1 g which crosses the abscissa at about 0.1 g and giving an impression that the g -value must be more than 0.1 g to give nystagmus in caloric tests.

If one extrapolates the figures in Table 1 for cold water syringing to values less than 1 g one arrives at Fig. 7. According to the theory here used the nystagmus intensity ought to be zero in conditions of weightlessness. That the extrapolation here arrives at an imaginary 0.5 g instead of zero is either due to the lack of sufficient material or to the want of absolute precision in the experiments—it is not possible to say which. One cannot rule out that it may also be an expression of the sluggishness which occurs in the whole reacting system during a caloric test so that with a gravitational field of less than about 0.1 g no nystagmus results from the caloric test. Thus 0.1 g is the liminal g value for nystagmus in the caloric test.

ZUSAMMENFASSUNG

Kalorische Prüfungen wurden an vier Versuchspersonen in einer grossen Zentrifuge bei 1 g , 1.25 g , 1.5 g , 1.8 g vorgenommen. Die Versuche haben gezeigt: 1) Kalorische Prüfungen werden durch die Stärke des Gravitationsfeldes beeinflusst; 2) die maximale Nystagmusintensität nimmt mit höheren g Werten zu; 3) diese Zunahme ist direkt proportional zu der Zunahme in der Stärke des Gravitationsfeldes; 4) die Latenzperiode nimmt mit höheren g Werten ab; 5) die Ergebnisse sind gleich bei kalt- und Heisswasserspülung. Es hat sich auch gezeigt, dass die Nystagmusdauer mit höheren g Werten zunimmt, aber das Material ist im Hinblick auf diesen Aspekt nicht vollständig. Eine Extrapolation der Ergebnisse in Richtung auf g Werte unter 1 deutet auf ein Verschwinden des Nystagmus im Zustand der Schwerelosigkeit, wobei der Schwellenwert für den Nystagmus etwa 0.1 g ist. Eine Versuchsreihe über die kalorische Prüfung und Bergewicht bei höheren g Werten ist geplant.

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MYRINGOPLASTY WITH A FREE GRAFT FROM THE EXTERNAL AUDITORY CANAL

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The conspicuous anatomical similarity between eardrum tissue and the skin of the auditory canal leads to the theoretical deduction that this skin should be an excellent material for myringoplasty. It might likewise be assumed that in view of the exceptional viability of such skin even on a poorly supplied graft bed myringoplasty could be done successfully with the use of a free graft.

In the hope of confirming these assumptions a series of operations was undertaken with the use of a reported method employing free grafts from the auditory canal. The series comprises 24 cases of major eardrum perforations which after the operation were subjected to regular follow ups with audiometric examinations and eardrum microscopy for at least three months. The findings indicate that genuine ingrowth of transplanted tissue occurs. More than 80 per cent of the cases showed healing of the eardrum. As regards the cases with intact ossicular chains hearing improvement was noted in approximately 80 per cent and normalization of the hearing in one half. These results serve to confirm the theoretical deduction that the skin of the auditory canal is an ideal substitute for eardrum tissue.

Aspects on skin grafts for myringoplasty

In cases with large eardrum defects regeneration from the persisting drum membrane is frequently so impaired as to preclude satisfactory healing even with the operative aid of scarification and covering of the perforation. The lamina propria as Lüscher has shown plays but little part in spontaneous healing and hence the resulting scar is flaccid. In total perforations healing is as a rule precluded because the regenerative centers of the eardrum have been eliminated. The eardrum skin has a germinal center in the area round the short process of the malleus and according to Stinson the proliferation there is so great that the tympanic membrane epithelium gradually spreads towards the external auditory canal.

With major perforations of the eardrum therefore adequate restitution of the membrane is possible only by actual ingrowth of transplanted tissue. The latter must be viable and must consist of tissue elements congruous with those of the eardrum. The drum membrane is of course composed of skin and mucosa which continue diffusely into the contiguous structures as well as an intermediate fibrous layer of collagenous fiber bundles arranged in radial and circular bands. Adjacent to the tympanic ring there is an additional element of elastic fibers which in the cartilaginous ring itself form a

thick elastic band. The true inelastic drum membrane is thus fixed in an elastic framework—a condition which is important for the acoustico dynamic function.

Ordinary skin which is generally used in myringoplasties contains little collagen but a substantial amount of elastic connective tissue as well as subcutaneous fat. Both the elastic tissue and the fat are to be regarded as elements useless for restitution of the drum membrane. In those parts of the facial skin which lie truly on the underlying cartilage or bone and are not subject to stretching both the elastic tissue and the fat content is less than that in other areas. Wullstein therefore uses skin from behind the external ear and the adjacent portion of the mastoid region. In this way he secures a graft which not only has little tendency to curl up but which can be obtained directly from the surgical wound by the retroauricular approach.

In the case of major eardrum perforations the grafts are largely dependent on nutrition from the periphery. From the graft bed the transplant must bridge a defect which in cases with total perforation amounts to approximately 60 sq. mm. The transplanted tissue must accordingly possess a substantial anastomosing vascular network with the ability to unite with that of the graft bed and thence supply even the central parts of the graft. To secure such a network it is customary to employ skin grafts with an adequate subcutaneous layer. Such full thickness skin grafts contain however an excess of nonspecific subcutaneous tissue which during healing extends towards the tympanic cavity and there gives rise to exudate accumulation with the attendant danger of synechia formation. With a sufficiently superficial and well anastomosed vascular network it would be possible to make the grafts thinner without danger of necrosis and if moreover the subcutis had a structure specific for the drum membrane the undesirable extension of the subcutaneous tissue could perhaps be reduced further.

Microanatomical resemblance of the eardrum to the skin in the auditory canal

There is one area of skin—i.e. the external auditory canal which satisfies all of the above mentioned criteria for a satisfactory graft and which in addition is in close proximity to the operative field. This skin has an epithelial layer congruous with that of the eardrum and like the latter is supplied by arteries from the periphery. A vascular plexus running from the deep auricular artery along the eardrum margin supplies both the external surface of the drum membrane and the skin of the auditory canal with an abundant superficial vascular network which extends to the external meatus before uniting with other vessels and is moreover relatively devoid of vascular communications with the underlying tissue. It is thus possible to undermine the soft tissues without appreciable bleeding as far as the eardrum margin whereas even slight laceration of the auditory canal epithelium results in profuse bleeding.

The subcutis of the external auditory canal is firm, fibrous and plentifully

supplied with collagenous fibers. The elastic fibers are very few in number and in this area there is no layer of fat. The skin of the auditory canal possesses lastly a good healing capacity and a marked tendency to take well even on poorly supplied graft beds—properties which Lempert utilized ten years ago in epithelialization of radical operation cavities with free grafts from the auditory canal.

Myringoplasty with pedicled graft from the auditory canal

In view of the above mentioned characteristics skin from the auditory canal seems to be eminently suited for grafting in myringoplasties. Indeed Frenchner (1957) reported a method involving the use of a pedicled flap from the auditory canal which together with underlying periosteum was brought up over the eardrum perforation. He found excellent healing with a membrane so closely resembling the eardrum tissue that he was inclined to attribute the closure of the perforation to ingrowth along the flap of tissue from the remaining part of the drum membrane. The excellent cosmetic results were supplemented in most cases by hearing improvements which Hjorth analyzed in a paper published the same year. In 1957 however Frenchner reported in a review of tympanoplasties that he had gradually gone over to the technically less complicated procedure of grafting by the Wullstein method whereby he had obtained similar hearing improvements but some what poorer cosmetic results.

It is more than likely that in Frenchner's cases genuine healing occurred. It is also probable that equally good results could be secured by employing free grafts from the auditory canal.















OWN INVESTIGATION WITH FREE GRAFT FROM THE AUDITORY CANAL

In the hope of confirming this view a series of myringoplasties with the use of such free grafts has been undertaken at the Ear Department of Danderyd Hospital since January 1960. For ears with dry perforations involving 50-100 per cent of the drum membrane Zollner I-II operations were the standard procedure regardless of the state of the ossicular chain. Closure of the perforation could be expected to result in hearing improvements in cases with intact ossicular chains; for the other cases additional surgery to improve the hearing was planned for a later stage. In this way some measure of homogeneity was secured in the series which was to be evaluated primarily with respect to the healing results.

Although some thirty patients have so far been operated on the following report includes only those with a postoperative follow up time of at least three months. The series consists of 24 patients—12 males and 12 females—ranging in age from 7 to 73.

The preoperative clinical investigation in each case included ear roentgenograms, audiometry, inspection of the eustachian tubes and microscopic examination of the eardrum. In the majority of cases prosthetic appliances were also tried. In no case was cholesteatoma present. One patient had

TABLE 1 Cases with normal ossicular chain (1-14)

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4 Female, 42 Op 7 4 1960 Results Perforation closed Hearing improved (normal)		11 Female, 51 Op 17 11 1960 Results Perforation closed Hearing improved	
5 Male, 11 Op 15 6 1960 Results Perforation closed Hearing not improved		12 Male, 19 Op 18 11 1960 Results Perforation closed Hearing improved (normal)	
6 Male, 53 Op 29 6 1960 Results Perforation closed Hearing improved		13 Female, 26 Op 23 11 1960 Results Perforation closed Hearing improved	
7 Male, 13 Op 12 7 1960 Results Partial necrosis in the flap Hearing not improved		14 Male, 32 Op 7 12 1960 Results Perforation closed Hearing improved (normal)	

blue in color thereafter assuming more and more the appearance of the original drum membrane. In many cases the healed membrane could not be distinguished from a normal eardrum even under high magnification.

When in rare cases, necrosis appeared in the graft, it was observed within the first month but did not thereafter increase in extent. The necrotic areas were dry and, at tubal catheterization, no secretion was voided through the perforation.

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chronic osteitis which led to excavation of the cellular system in connection with the myringoplasty.

Operative method

The operations were performed as a rule under local anesthesia with the use of 10 cc xylocaine carbaine and an injection technique according to Zollner. Only in a few cases (children) general anesthesia was given.

Following an endaural incision according to Lemperth the entire posterior portion of the soft auditory canal was undermined as far as the eardrum margin after which a longitudinal incision was made through all soft tissues, the incision being carried both directly upwards and directly downwards in the auditory canal. The skin was then removed after being cut at the eardrum margin. The piece of skin thus obtained had a surface area of 1.5 by 2 cm and decreased in thickness towards the inner border where it was as thin as the drum membrane. It was immersed in physiologic salt solution pending preparation. Visualization of the eardrum region was more extensive after removal of the canal skin.

For sealing of persistent drum membrane the epithelium was incised round the perforation at a distance of 1-2 mm from its edge after which superficial radial incisions were made. The cutaneous layer was then scaled with a desquamator in the direction of the perforation so that the flaps after trimming could be used to partially cover the opening. The perforation was thus reduced in size and a larger bed was obtained for the graft. The remainder of the pars tensa, the pars flaccida and the limbus were carefully freed of epithelium. The skin in the anterior half of the internal auditory canal was undermined as far as the eardrum margin and folded upwards so as to expose an area of bone 4-5 mm in size along the drum membrane.

The graft from the auditory canal was then thinned into a flexible transplant. Since this showed no tendency to curl it was applied without much difficulty, the thin inner border being placed against the exposed bone anteriorly after which the skin of the auditory canal was so reflected as to overlap the graft along a stretch a few millimeters in length. The anterior middle portion was placed in contact with the drum membrane itself and the outer border covered the posteriormost part of the exposed area of bone. All blood was aspirated from the tympanic cavity prior to application of the graft, the cavity subsequently being left empty. The graft was kept in position by a fine terracortril pack after which the entire auditory canal was filled with another thicker terracortril pack. The exposed bone was left without a skin covering. The end of a finger stall was placed in the external meatus and the skin incision sutured (Figs. 1).

Postoperative follow up

The patients were allowed up the day after operation and discharged after one week. The finger stall was removed on the fourth day, the external

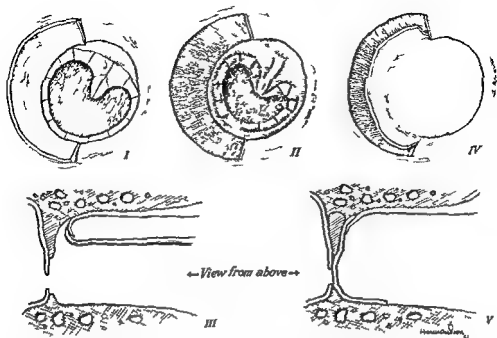
















FIG. 1 I Entire posterior portion of the soft and tory canal undermined. Incisions made in the epithelium of the drum. II Skin graft removed. Sealing of the drum. Epithelium folded in at the edges of the perforation. III Skin in the anterior part undermined at limbus. IV and V Skin graft in position.

canal pack after one week and the inner pack after two weeks. In each case the postoperative course was uneventful and neither hyperthermia nor pain was noted. Only a single ear—the one concurrently subjected to radical surgery—showed a slight discharge; the others remained dry. The patients thereafter attended the hospital for regular follow-ups including tubal catheterization, microscopic examination of the eardrum, and audiometric tests.

Tubal catheterization was first undertaken two weeks after the operation, then once weekly for some weeks, and subsequently in connection with monthly follow-ups. Even at the initial examination the eustachian tube in all cases was found to be patent. Although occasional rales were detected at auscultation, they had disappeared after a few catheterizations.

Graft healing was studied by microscopic examinations of the eardrum. On removal of the terracortril pack after two weeks, the graft was pink in color, slightly edematous, and without discernible details. Two weeks later it was covered by a grayish brown, slightly butyraceous film which was readily detached. Beneath this film the graft had a fatty and somewhat moist appearance, and high magnification disclosed numerous pink points from which extremely fine serpentine vessels radiated. After a further two weeks the surface was epithelialized and the graft dry. By this time the examination revealed numerous vascular arcs indicative of anastomotic processes. Some months after the operation the graft was thinner and gray.

TABLE 1 Cases with normal ossicular chain (1-14)











1 Male, 43 Op 17 2 1960 <i>Results</i> Perforation closed Hearing improved		8 Female, 15 Op 17 8 1960 <i>Results</i> Perforation closed Hearing improved (normal)	
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4 Female, 42 Op 7 4 1960 <i>Results</i> Perforation closed Hearing improved (normal)		11 Female, 51 Op 17 11 1960 <i>Results</i> Perforation closed Hearing improved	
5 Male, 11 Op 15 6 1960 <i>Results</i> Perforation closed Hearing not improved		12 Male, 19 Op 18 11 1960 <i>Results</i> Perforation closed Hearing improved (normal)	
6 Male, 53 Op 29 6 1960 <i>Results</i> Perforation closed Hearing improved		13 Female, 26 Op 23 11 1960 <i>Results</i> Perforation closed Hearing improved	
7 Male, 13 Op 12 7 1960 <i>Results</i> Partial necrosis in the flap Hearing not improved		14 Male, 32 Op 7 12 1960 <i>Results</i> Perforation closed Hearing improved (normal)	

blue in color, thereafter assuming more and more the appearance of the original drum membrane. In many cases the healed membrane could not be distinguished from a normal eardrum even under high magnification.

When, in rare cases, necrosis appeared in the graft, it was observed within the first month but did not thereafter increase in extent. The necrotic areas were dry and, at tubal catheterization, no secretion was voided through the perforation.

The grafts from the auditory canal contained hairs. These were usually shed after six to eight weeks, by which time the eardrum showed every sign of vitality. In some cases, however, hairs persisted at the periphery

TABLE 2 Cases with ossicular chain broken (15-24)
No or little improvement in hearing

15 Male 41 Op 23 3 1960 Results Total necrosis in the flap		20 Male 47 Op 24 8 1960 Results Partial necrosis in the flap	
16 Female 34 Op 18 5 1960 Results Perforation closed		21 Male 7 Op 25 8 1960 Results Perforation closed	
17 Female 34 Op 3 6 1960 Results Perforation closed		22 Female 47 Op 9 9 1960 Results Perforation closed	
18 Male 30 Op 10 6 1960 Results Perforation closed		23 Female 45 Op 14 9 1960 Results Perforation closed	
19 Female 13 Op 18 8 1960 Results Perforation closed		24 Female 44 Op 18 1 1961 Results Perforation closed	

and appeared to have grown thicker resembling long black ink marks along the eardrum margin















The auditory canal returned to its normal appearance within a very short time even though half of it had been removed at the operation. The skin defect was covered by a free graft according to Wulstein after the first six operations but not after subsequent ones. Despite the lack of a covering graft epithelialization occurred very swiftly from the persistent skin with no tendency to stenosis.

The 24 cases are detailed in Tables 1-2. The grafts in four cases developed necrosis—total in one (Case 15) and partial in three (Cases 7, 9, 20). Healing took place in 20 patients i.e. more than 80 per cent of the series.

It was possible to study the hearing improvement in those cases which had intact ossicular chains prior to operation i.e. 14 patients (Tables 1 and 11). A hearing improvement of 15 db or more was recorded in 11 cases in seven of which the hearing was in fact normalized. The postoperative hearing improvement accordingly amounted to about 80 per cent in this series and normal hearing was obtained in 50 per cent.

The investigation presented here serves to confirm the theoretical deduction that skin from the auditory canal is an ideal substitute for eardrum tissue. The operative method with the use of free grafts from the auditory canal evidently enhances the possibilities for normalization of the eardrum condition in otherwise difficult cases of perforation.

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1 Male, 43 Op 17 2 1960 Results Perforation closed Hearing Improved		8 Female 15 Op 17 8 1960 Results Perforation closed Hearing Improved (normal)	
2 Male, 38 Op 2 3 1960 Results Perforation closed. Hearing Improved (normal) See 1206/60		9 Male 38 Op 27 10 1960 Results Small necrosis in the flap Hearing not improved See Case 11	
3 Female, 38 Op 6 4 1960 Results Perforation closed Hearing improved (normal)		10 Female 15 Op 2 11 1960 Results Perforation closed Hearing improved (normal)	
4 Female, 42 Op 7 4 1960 Results Perforation closed Hearing improved (normal)		11 Female 51 Op 17 11 1960 Results Perforation closed. Hearing improved	
5 Male, 11 Op 15 11 1960 Results Perforation closed Hearing not improved		12 Male, 19 Op 18 11 1960 Results Perforation closed Hearing improved (normal)	
6 Male, 53 Op 29 6 1960 Results Perforation closed. Hearing improved		13 Female 26 Op 23 11 1960 Results Perforation closed. Hearing improved	
7 Male, 13 Op 12 7 1960 Results Partial necrosis in the flap Hearing not improved		14 Male, 32 Op 7 12 1960 Results Perforation closed Hearing improved (normal)	

blue in color thereafter assuming more and more the appearance of the original drum membrane. In many cases the healed membrane could not be distinguished from a normal eardrum even under high magnification.

When in rare cases, necrosis appeared in the graft, it was observed within the first month but did not thereafter increase in extent. The necrotic areas were dry and at tubal catheterization no secretion was voided through the perforation.

The grafts from the auditory canal contained hairs. These were usually shed after six to eight weeks, by which time the eardrum showed every sign of vitality. In some cases, however, hairs persisted at the periphery

DIFFERENT INFLUENCE ON VESTIBULAR FUNCTION FOLLOWING UNILATERAL LABYRINTHECTOMY

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Central compensation following unilateral labyrinthectomy has been studied and discussed in an earlier paper (Fluur 1960). By depressing the activity in the intact labyrinth by means of cold water irrigation it was possible to demonstrate a gradual return of the normal function via compensatory activation of the vestibular nuclei on the labyrinthectomized side.

Bárány (1906-1907) conducted rotatory tests on patients bereft of one labyrinth due to acute labyrinthitis. He found far greater sensitivity to ampullopetal than to ampullofugal endolymph currents and sought to explain the phenomenon on the basis of Iwald's (1892) view that the horizontal semicircular ducts had a unidirectional sensitivity which could come into play only after loss of one labyrinth. Cawthorne, Fitzgerald & Hallpike (1942) reported caloric tests of patients who had undergone unilateral labyrinthectomy; they too demonstrated increased sensitivity to ampullopetal endolymph currents. They termed this phenomenon 'directional preponderance' and attributed it to conversion of the normal bi-directional sensitivity into an abnormal unidirectional type.

Since different authors have placed different interpretations on their results it has been considered worth while to investigate the possible effects of unilateral labyrinthectomy on the function of the intact labyrinth.

METHOD

Five persons with Menière's disease who were to undergo unilateral labyrinthine destruction were examined preoperatively with caloric tests according to the technique described by Fitzgerald & Hallpike (1942). Nystagmus was concurrently recorded electro-nystagmographically. Post-operatively the responses of the intact ears to caloric tests were examined regularly each month for periods of up to half a year.

A sixth patient with acute otitis developed labyrinthitis which abolished the function of one ear. He was subjected to caloric tests and nystagmography daily for fourteen days then monthly for six months.

RESULTS

Reliable evaluation of the results was not possible until the acute nystagmus due to the labyrinthine destruction had subsided (i.e. after 2-3 wks).

one month. Slight nystagmus towards the intact side was still observed after that time in three of the patients on recording with the eyes closed. It did not however impede a study of calorically induced nystagmus which was based on frequency and duration.

All of the labyrinthectomized patients showed on hot water irrigation a postoperative increase in the frequency and duration of nystagmus. It was not possible to date the onset of this increase with assurance. It had probably begun soon after operation and was thus masked by the spontaneous nystagmus. Four patients exhibited one month after operation a marked increase in the frequency and duration of nystagmus after hot water irrigation the duration exceeding the preoperative values by 30 to 95 seconds. The fifth patient had only a minor prolongation amounting to 10 seconds. All five still showed after two months a duration increase of between 10 and 40 seconds. When three months had elapsed an increase of 25-30 seconds persisted in two patients, the preoperative values had been restored in another two and in the fifth the duration was 10 seconds shorter than it had been preoperatively. During the remaining three months the durations fluctuated a little on either side of the initial values (Fig. 1).

Since the sixth patient had not undergone caloric tests before the onset of labyrinthitis no comparisons with the function prior thereto were possible. The regular examinations following labyrinthectomy indicated however a distinct fall in the duration of nystagmus induced by hot water irrigation.

DISCUSSION

The investigations has shown that a person bereft of one labyrinth exhibits an absolute increase in the duration of nystagmus induced by irrigation with water at a temperature exceeding 37°C.

The heightened sensitivity to ampullopetal endolymph currents in an intact horizontal semicircular canal has been demonstrable only after loss of the contralateral labyrinth. Apparently therefore labyrinthectomy not only deprives the ipsilateral nuclei of their spontaneous activity but also promotes facilitation on the contralateral side—peripherally in the labyrinth or centrally in its nuclei (Fig. 2). Two important questions immediately arise. First, what pathways does this influence follow—direct communications between the two sides or more circuitous routes? Secondly is the facilitation due to loss of an inhibitory influence or to a genuine increase of activity in the remaining vestibular apparatus? Since these questions are closely interrelated they can scarcely be discussed separately but will be treated together.

Several authors have sought to establish whether direct and mutual communications exist between the two vestibular organs. Eddler (1914) and Ingvar (1918) found direct pathways between the vestibular nerve and the contralateral vestibular nuclei. Gray (1926) claimed by section of the vestibular nerve to have demonstrated such direct fibers but was unable

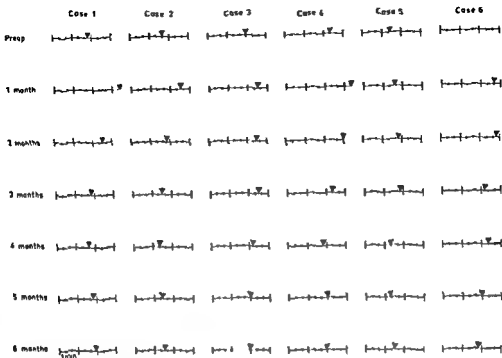


FIG. 1. Durations of nystagmus after hot water irrigation of the normal ear in patients with monolateral destruction of one labyrinth.

to preclude the possibility that Deiter's nucleus had been traumatized with consequent secondary degeneration. Rasmussen (1932) and Walberg Bowsher & Brodal (1958) conducted similar experiments but they flatly discount the idea that direct fibers pass from the vestibular nerve to the contralateral nuclei. Several authors (Rasmussen 1932, Ferraro, Pacella & Barrera 1940) have nevertheless identified occasional secondary commissural fibers passing between the nuclei of the respective sides. In the opinion of other workers these fibers do not belong to the vestibular nerve but stem either from the facial nerve (Kohnstamm 1902, Kaida 1929) or from the olivary peduncle (Papez 1930).

The anatomical investigations have thus yielded conflicting results. The current view appears to be, however, that there are no direct fibers between the peripheral labyrinth and the contralateral vestibular nuclei but that a few commissural fibers may possibly connect the nuclei of the two sides.

Somewhat varying data have emerged also from the neurophysiological investigations. Gernandt & Hultin (1952) obtained on rotation no response from the nuclei of the labyrinthectomized side but unchanged fully normal responses from the nuclei of the intact side. In contrast, De Vito, Brusa & Arduini (1956) observed on cathodal polarization of the peripheral labyrinth an increase in the activity of the ipsilateral Deiter's nucleus in 58 of 63 leads at the same time they recorded in the contralateral nucleus an increase in light and blocking in 22 of 39 units. After subsequent decerebellar

tion of the animals the authors found an increase in eight and blocking in six of 16 units. No recording was done after unilateral labyrinthectomy. Their findings do not point to the existence of direct pathways between the two nuclei. The results could be taken to imply rather that impulses mediated via the cerebellum exert an inhibitory influence on the contralateral nuclei provided there is an increase in the activity of the stimulated labyrinth, i.e. a situation corresponding to ampullopetal endolymph flow in the horizontal semicircular canal.

There is no anatomical or neurophysiological evidence that the facilitation of intact vestibular organs demonstrated in this investigation stemmed from a reciprocal innervation mechanism directly connecting the two nuclei. The nervous communications observed by some authors may thus be assumed to possess other functions.

The influence on the contralateral nuclei which unquestionably follows labyrinthectomy appears rather to be mediated via indirect secondary pathways. There are several efferent pathways to the vestibular nuclei. Some stem from the oculomotor center, where Lorente de No (1933) and Pompiano & Wallberg (1957) observed descending fibers passing from the nucleus interstitialis of Cajal to the ipsilateral medial vestibular nucleus; others start from the reticular formation—both at its mesencephalic level (Langworthy 1928) and at its pontine (Mettler 1944) and medullary levels (Langworthy 1929, Mettler 1944, Beusekom 1955) and yet others issue from the cerebellum which has long been known to be significantly interrelated with the vestibular nuclei. Common to these secondary centers is the fact that each of them constitutes a functional unit with very close interaction between the respective sides. The activity of the two sides is well balanced under resting conditions, yet exogenous factors may trigger the reflex mechanism and thus rise to either facilitatory or inhibitory effects on other equally well balanced centers.

Since the fibers stemming from the nucleus interstitialis of Cajal appear to be concerned in vertical and rotary movements of the eyes (Szentágothai 1943) they may be disregarded here for in this investigation only horizontal nystagmus was recorded. Of greater importance are the pathways from the mesencephalic reticular formation from which horizontal eye movements are controlled (Szentágothai 1943) at least insofar as they are facilitatory. The brain stem reticular formation represents one of the most important integrating structures in the central nervous system. Investigations by Magoun & Rhines (1946) and by Rhines & Magoun (1946) have established that the reticular formation contains both facilitatory and inhibitory regions the reciprocal action of which is exceedingly well balanced. All motor activity is associated likewise with very close interplay between the reticular formation and the vestibular apparatus. This interaction is probably controlled both at peripheral and at central levels by facilitatory as well as inhibitory pathways which connect the two centers and thus promote their reciprocal activity as demonstrated by Gerbardin & Thulin (1952, 1953).

With unilateral loss of labyrinthine function the facilitatory influence on

LABYRINTHINE FUNCTION
ACUTE (UNCOMPENSATED) MONOLATERAL
DESTRUCTION

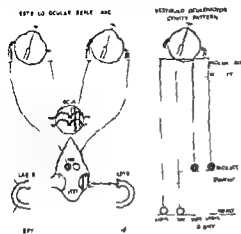


Fig. 2

LABYRINTHINE FUNCTION
COMPENSATED MONOLATERAL DESTRUCTION
HOT WATER IN RIGHT EAR

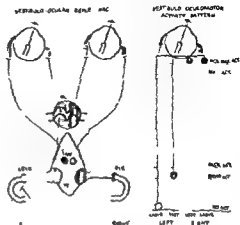


FIG 3

110 2 Labyrinthine function acule (uncompensate) monolateral destruction  = no activity

☐ Isotonic & spontaneous activity

110. 3 Labyrinthine function compensated monolateral destruction hot water in right ear

 = spontaneous activity increased and facilitated activity

the contralateral vestibular apparatus might be exerted via either of two possible processes that would ultimately have the same effect—increased spontaneous activity. Either release occurs from an inhibitory mechanism in the vestibular nucleus which via the reticular formation acts upon the contralateral homonymous nucleus or else the compensatory process described in an earlier paper (Fluur 1960) also affects the intact vestibular organ. In the latter case there would be a

The second possibility postulates however a complex linking system in which both the facilitatory and the inhibitory regions would have to be involved. Each of the two possibilities mentioned above is conceivable.

The cerebellum is also one of the most important organs in the body for co-ordination and integration of motor activity. Its communications with the vestibular organs are so extensive that certain centers in the cerebellum are rightly regarded as direct outgrowths from the vestibular nuclei. Histological investigations have revealed the existence of both vestibulo cerebellar and cerebello vestibular pathways (Brodal & Torvik 1917; Cohen, Chambers & Sprague 1918; among others) Bauer & Leidler (1912) and Pollock & Davis (1927) assumed from their investigations that the cerebellum exerts an inhibitory action upon vestibular centers. This has since been confirmed

by a number of workers (for further references see Dow & Moruzzi 1958). With loss of the inhibitory influence after unilateral labyrinthectomy facilitatory impulses are released from the caudal pole of the fastigial nucleus and then act upon the contralateral vestibular nuclei via the uncinate fasciculus of Russell (Moruzzi & Pomperino 1957; Carpenter 1959). Numerous anatomical and neurophysiological observations suggest therefore that the facilitation which influences the intact vestibular apparatus after unilateral labyrinthectomy is mediated via the cerebellum.

The demonstrated facilitation may affect the vestibular apparatus at different levels. An increase of the spontaneous activity in the vestibular nuclei is conceivable. Equally possible however is that the peripheral labyrinth contains an efferent inhibitory system of the same type as that in the cochlea (Galambos 1956) and that its activity ceases with removal of the contralateral labyrinth. Studies on human subjects could hardly suffice however, to establish which of these possibilities is correct; detailed neurophysiological studies on suitable laboratory animals would be required.

CONCLUSIONS

1. The increase in the duration of nystagmus which follows unilateral labyrinthectomy can be demonstrated on ampullopetal endolymph flow in the horizontal semicircular canal of the intact labyrinth is due not to uni- or bi-directional sensitivity of the peripheral labyrinth but to facilitation of efferent impulses reaching the vestibular apparatus from supravestibular centers in the cerebellum and reticular formation and possibly also to afferent impulses from spinal centers (Fig. 3).

2. Depression of the peripheral activity by cold water irrigation on patients with normal vestibular organs facilitates the contralateral activity; augmentation by hot water irrigation inhibits it. This accounts for the longer duration of nystagmus on cold water than on hot water irrigation in caloric tests of normals.

3. Objections to Wald's second law cannot be raised on the basis of findings in monolabyrinthine patients whose response to hot water irrigation is dependent upon postoperative the facilitatory effect of supravestibular centers on the vestibular organ.

4. Directional preponderance is a sign of activity imbalance between the central nuclei.

SUMMARY

Six unilaterally labyrinthectomized patients were investigated with warm water irrigation of the sound ear. All showed a postoperative increase in the duration and frequency of nystagmus. This increase was attributed to the fact that each of the two labyrinths normally exerts an inhibitory influence on the others' activity. Following a lesion of one labyrinth this inhibitory effect disappears and instead there occurs facilitation of the activity of the sound vestibular organ. The physical mechanism underlying this facilitation is discussed.

ZUSAMMENFASSUNG

Man untersuchte sechs unilateral labyrinthectomierte Patienten mit Warmwasserspülung des gesunden Ohres. Sämtliche zeigten eine postoperative Steigerung der Duration und Geschwindigkeit des Nystagmus. Von dieser Steigerung nahm man an, dass sie darauf beruhte, dass die beiden Labyrinthe normal einen hemmenden Einfluss auf ihre gegenseitige Aktivität haben. Bei einem einseitigen Labyrinthschaden fällt diese Hemmung fort und statt dessen tritt eine Facilitierung der Aktivität des frischen Vestibularisorgans ein. Der physiologische Mechanismus dieser Facilitierung wurde diskutiert.

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MEDIASTINOSCOPY—A NEW FIELD FOR BRONCHOLOGISTS

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Examination of the anterior mediastinum using Carlens' middle line approach is described and experience reported on 87 patients. Special emphasis is laid on various points in the procedure which require special care and on certain steps which facilitate the safe removal of biopsy specimens. The macroscopic characteristics of carcinomatous metastases, vascular anomalies, cysts and sarcoid lesions are described. No complications occurred in the course of mediastinoscopy.

The direct examination of the anterior mediastinum has become possible lately through the ingenious approach of Carlens, who described the method in 1959 reporting his experience on over 100 patients. Briefly stated, this operation gives access to the mediastinum through a skin incision in the jugular fossa; the mediastinoscope is introduced through the deep neck fascia to the ventral surface of the trachea which it follows down to the bifurcation and along both main bronchi.

Mediastinoscopy proved a great step forward in the examination of patients suspected of mediastinal involvement, either of a carcinomatous process, a primary mediastinal tumour, or some systemic disease of the lymph nodes (Seppälä 1959, 1960; Palva 1960, 1961; Palva & Vuokari 1961). The examination readily discloses a tumor pathology and biopsies taken from suspicious nodes give a definite histological diagnosis. The procedure in itself is well tolerated by the patients, who need no extra time for convalescence; in fact, if necessary, the patients can leave the hospital on the day following the operation.

The technical details have been discussed earlier by Carlens (1959), Palva & Vuokari (1961) and Palva (1960, 1961) so comprehensively that little need be added, and the reader is referred to those earlier papers. The method is not very difficult to acquire if the surgeon is well accustomed to both neck surgery and endoscopic procedures, and by nature willing to proceed with gentle hands. It is not a procedure to be done by assistants or residents but by experienced specialists with either bronchologic or thoracic surgery training.

The mediastinoscope has to pass many vital structures on its way down to the bifurcation and the beginner will be very wise to refresh his memory of the upper mediastinal anatomy in an autopsy room. The work on patients can then be begun much more safely.

Read at the Meeting of the Finnish Otolaryngological Society, Helsinki, February 11, 1961.

In this paper I propose to outline some points in the procedure which may cause difficulties at the beginning and I will also report my present experience with mediastinoscopy

Important Aspects in Mediastinoscopy

After the incision in the jugular fossa, the trachea is freed below the thyroid isthmus. The venous plexus is well developed in this area and the veins may be greatly distended especially in patients suffering from pulmonary emphysema. Under direct vision one or two veins may be ligated and cut but very often the trachea can be freed without bleeding from the vessels. If any one of the veins is cut brisk bleeding ensues which is best controlled with firm pressure. The bleeding vein is then sought, clamped and tied. It is not advisable to proceed lateral to the trachea as the recurrent nerves might be injured.

My series includes two cases in which because of vascular anomaly the anonymous artery was up in the neck over the trachea. Before the tissues are spread bluntly with scissors it is advisable to feel with the index finger that there are no pulsating structures on top of the trachea which if unnoticed would give rise to serious bleeding.

If the trachea deviates so much to one side that it goes down not in the sternal notch but below the clavicle it is nearly impossible to introduce the mediastinoscope through this narrow slit without total compression of the tracheal lumen. This fact will be clearly visible beforehand in A-P roentgenograms and mediastinoscopy is then best cancelled.

Before preparing the way with the index finger into the mediastinum one must be definitely through the deep neck fascia on top of the trachea. If one tries to force one's way into the mediastinum above the deep fascia one enters a wrong space where separation of the tissues without trauma is impossible and complications are bound to occur. On the other hand the tracheal wall should always be handled very gently. Otherwise there will be an annoying postoperative tracheitis following small lacerations of the tracheal mucosa caused by the intubation tube.

The mediastinal arteries may show abnormalities in many areas. These most frequently become apparent to the mediastinoscopist in the form of aneurysms in one of the major arteries. If the aneurysm is situated in the aorta itself it generally fills entirely the space between itself and the trachea and the finger has to displace the posterior aneurysmal wall forward in order to explore the lower areas. In these cases the index finger, feeling the pulsating structure, is the best instrument and the one to be exclusively used. I once made a puncture and aspiration of a small sized aortic aneurysm in my early cases but this is entirely superfluous and might be dangerous should the aneurysmal wall be distended and thin. Other aneurysms that may occur are those of the anonymous artery or subclavian artery. Once the finger defines their pulsation, their size and location, all that is necessary for diagnosis is known.



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FIG. 1 Normal mediastinal lymph node. FIG. 2 Black, antracotic lymph node. FIG. 3 Large, glandular mass in sarcoidosis. FIG. 4 Large blackish lymph node with metastases of microcellular carcinoma. FIG. 5 Mediastinal extension of spinocellular carcinoma. FIG. 6 Large aortic aneurysm.

Providing that the arteries are normal the mediastinoscope slides easily below the aortic arch towards the bifurcation. The points to be closely observed in this area are the azygos vein on the right side and the pulmonary vessels on the left. The azygos vein is not always seen if there is enough glandular tissue close to the lower part of trachea and bifurcation. However, in some cases one has to make a search of glands over the right main bronchus above the take off of the upper lobe bronchi and here the azygos vein is often noted. Its wall shows the same bluish colouring as many lymph nodes in the area but it is always longitudinal in structure and can generally be interpreted as vein. In one case I punctured it a vein can readily be distinguished from a gland on puncture since the former offers considerable resistance to the needle whereas the latter is entered by the needle without any resistance. On withdrawal of the needle there is some bleeding from a vein and one to two minutes compression is required for the bleeding to stop. From a gland there is no bleeding although a drop of blood may be seen at the puncture point.

The pulmonary vessels on the right side pass below the upper lobe branches the most distal point of mediastinoscopic exploration except in the case of some smaller blood vessels the right side does not present any major bleeding sources. On the left the situation is different as the pulmonary artery and vein cross the main bronchus proximal to the upper lobe bronchi. The arterial wall especially is quite fragile and does not tolerate any tearing forces applied to it. It is prudent to work on the left mainly on the superior side of the bronchus and only for a short distance on its ventral surface. The glands generally are in the superior tracheobronchial corner and here the pulmonary vessels are safely avoided.

In certain cases of cardiac hypertrophy the heart walls may extend high up into the mediastinum. However these cases are generally diagnosed by other means and mediastinoscopy is not needed for the purpose.

In addition to the recurrent nerves in the neck two other points should be observed in order not to injure the nerves. On the left the recurrent nerve can sometimes be seen as it descends under the aortic arch to the lateral side of the trachea. This is the case especially if there is some expansive process which displaces the tissues towards the right side. On the right the vagus nerve is sometimes seen descending downward close to the level of the bifurcation. The nerve trunks are of such size that they are recognized without any trouble. The left vagus and both phrenic nerves run so much above the mediastinoscopy level that they cannot be injured in the process.

When the adherent tissues are separated with the Carlen's tissue dissector, one can sometimes see a few air bubbles entering the mediastinum. The same may occur when a biopsy is taken from glands or tumor tissue directly continuous with the lung. The bubbles come from the parenchyma with pleural adhesions around and need not disconcert the operator, when the place is sealed with a sheet of gelfoam after completion of the mediastinoscopy no sequelae are observed. I have not seen a pneumothorax develop in

any of my cases and neither Carlens nor Seppili have reported this complication in their respective studies.

If bleeding occurs from the areas of dissection or of biopsy, it is generally easily controlled by slight pressure with a small dry tampon. Adrenalin is best avoided, as it can affect the circulation of these generally elderly people because of its rapid absorption from the loose mediastinal tissues. Application of a neomycin soaked slice of gelfoam will arrest any minor bleeding occurring from the operative field.

If the mediastinal carcinomatous masses involve large vessels, then a bleeding may occur that is clearly uncontrollable. This occurred in one case of Seppili's series (1961) in which there was bleeding from a large carcinomatous infiltration round the azygos vein. In the case of major bleedings temporarily but not permanently controllable with tamponade, thoracotomy and ligation of the blood vessels should be done immediately after mediastinoscopy.

Comment on Present Material

As pointed out earlier, mediastinoscopy aims primarily at gaining additional information about the operability of patients with pulmonary carcinoma, as well as at providing supplementary data for differential diagnosis. The results obtained in each material obviously depend much on this pre-operative selection, and it does not seem worth while to dwell upon statistics in great detail.

Among the 87 cases in this material there were 17 in which the final diagnosis was pulmonary carcinoma. Of these 19 were histologically verified only at mediastinoscopy, the primary tumor being outside the visual field at bronchoscopy and the loose cells in the Papanicolaou stain being repeatedly negative. There were several instances showing suspicious cells by the Papanicolaou method and carcinomatous infiltration of the mediastinal lymph nodes on biopsy.

Valuation of operability depends greatly on the thoracic surgeon, and we have agreed upon certain indexes of operability in the mediastinoscopic reports. Thus firm nodes with carcinoma occurring bilaterally are a definite contraindication to surgery, as well as larger carcinomatous masses around the bifurcation. Also, in the presence of direct extension of tumor tissue into the mediastinum, an operation is no longer justified. If the nodes involved are solitary, non-adherent to their surroundings and appear well resectable with the lung, then the decision rests mainly on functional studies of the lungs and the patient's general condition. This latter naturally also applies to the cases in which the mediastinal lymph glands have shown no evidence of carcinoma and the patient seems well operable from that point of view.

The series included 49 cases in which differential diagnosis between different diseases was attempted with the aid of mediastinoscopy. Of these 19 proved to be cases of carcinoma as discussed above. In 8 cases the diagnosis became satisfactory, in 4 vascular disease, in 3 a mediastinal cyst, while

in 7 cases the biopsies showed tuberculous involvement of the mediastinal lymph nodes. In 8 instances the pathological reports indicated only non-specific antracotic changes and no definite diagnosis could be established. These latter cases consist of various groups in which the tentative diagnoses range from sequelae of pleurisy or pneumonia to suspected carcinoma.

Mediastinoscopic diagnosis naturally rests mainly on histological examination of the removed biopsy specimens. In the majority of cases with carcinomatous metastases the nodes are hard on palpation and the diagnosis is obvious even clinically. However, the palpating finger may also in many cases encounter soft glands—this applies especially to microcellular carcinoma—and it is the pathologist who establishes the diagnosis. In squamous cell carcinoma the nodes are always hard on palpation.

Vascular anomalies in the arteries are best diagnosed with the palpating finger and require no further steps except for inspection of the area. In the case of a cyst diagnostic puncture and aspiration is indicated if there is a suspicion of malignancy, a biopsy specimen should also be removed.

While tuberculous glandular involvement does not in my experience show any microscopically identifiable characteristics, sarcoidosis presents typical features (Palva 1960, 1961). Active sarcoid glands are proliferating abundant violet coloured masses without the blackish tinge characteristic of normal mediastinal lymphnodes. When dissected free with gauze strips, dissectors or suction tips they bleed diffusely but not to a great extent. The biopsy pieces come off easily and the cut surface of the gland shows an even greyish cellular structure without the black smooth features of ordinary nodes.

In those cases of sarcoidosis in which the glands are at a stage of fibrosis and healing they generally show a distinctly bluish colouring always without the black features of normal antracotic nodes. They may be quite large, several centimeters in diameter, somewhat firmer to touch and not as easily bleeding as the glands in the active proliferating stage. In inactive stages one can sometimes see smaller nodes of normal appearance among the sarcoid glands.

Complications

In this series of 87 cases there has not been a single complication that might be ascribed to the mediastinoscopic procedure and the patients have been up and about on the first postoperative day. There was one exception in which a coronary thrombosis developed shortly after removal of the intubation tube and recovery of the patient from anaesthesia. As described earlier (Palva & Vukari 1961) this patient was successfully resuscitated.

Performed on the lines described above in the technical notes, mediastinoscopy thus seems to be an operation associated with little risk and very slight morbidity. The advantages gained from a direct examination of the anterior mediastinum on the other hand are so great and definite in selected cases that mediastinoscopy should be widely adopted in various thoracic



clinics for evaluation of those cases in which additional data are needed for evaluation of operability or differential diagnosis

ZUSAMMENFASSUNG

Die Untersuchung des vorderen Mediastinum mit Hilfe von Carlens Mittellineneingriff (middle line approach) bei 78 Patienten wird beschrieben und die dabei gemachten Erfahrungen werden besprochen. Verschiedene, besondere Aufmerksamkeit heischende Punkte des Verfahrens und gewisse Schritte, welche die gefahrlose Entnahme von Biopsieproben erleichtern, werden besonders hervorgehoben. Die makroskopischen Merkmale karzinomer Metastasen, vaskulärer Anomalien, Cysten und sarkalder Läsionen werden beschrieben. Keine Komplikationen traten im Lauf der Mediastinoskopie auf.

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GLANDULAR STRUCTURES IN THE PLANUM SEMILUNATUM

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At the base of the crista ampullaris can be found some epithelial formations that have been interpreted as acinous glands

In a recent work I have remarked that in the planum semilunatum can be distinguished four different zones: the zone of the wings, of the pavement of the bands, of the crista and the reticular zone. This last assumes topographic relations with all the others, so that many points of transition exist, besides. In the above mentioned work, I have emphasized that the reticular zone seems constituted of cells of two types, variously arranged so that in one tract it reminds one a glandular tissue. I thought it useful, therefore, to carry out a particular histological research the results of which seem important, both from a purely anatomical point of view and with regard to the functional interpretation.

The portion of planum semilunatum on examination corresponds to the two sides of the base of the superior crista ampullaris, sectioned according to different planes, in order to obtain the best evidence of the local epithelium both at the surface and deeply in.

Fig. 1 shows the superior crista of rabbit in an oblique projection, at the point in which the epithelium of the crista is in relation to that of the pavement of the planum one can clearly distinguish a cellular agglomeration, which sticks out and looks like a papilla. This structure, when magnified (Fig. 2) appears constituted of epithelial cells disposed round a central clear space so as to form a kind of corolla.

The epithelium of another area of the base of the crista, in connection with the reticular zone when seen in a cross section and magnified (Fig. 3) appears pluristratified with an irregular surface, while the deep stratum gets into the connective tissue of the crista, in the same way as the dermical papillae. The cells are disposed in different ways and seem collected in groups: some are incompletely separated by thin and irregular spaces containing a substance that reduces the osmic acid.

In the whole cellular polymorphous agglomerations are traceable, separated from one another whose volume increases, proceeding from the sensory epithelium towards the base of the crista.

Fig. 4 shows one of these structures which is puriform and run over by a central large duct with irregular walls, that extends from the ampullar



FIG. 1. Oblique section of crista. 1, epithelial papilla. 2, pavement of the plinum semilunatum.



FIG. 2. Detail of Fig. 1. Papilla formed by epithelial cells surrounding a clear space.



FIG. 3. Structural aspect of the epithelium of the base of the crista.



FIG. 4 The epithelial structure as seen at Fig. 3 but magnified

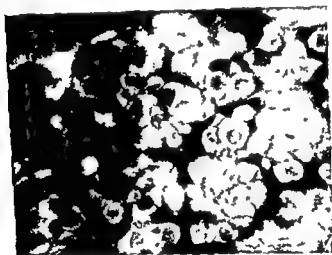


FIG. 5 Structure of the reticular zone near the base of the crista

cavity where it opens like an estuary up to the basal membrane. By means of convenient focal variations the duct is seen to branch out into smaller ones some of which reach the basal membrane.

The protoplasm of the cells constituting these structures contains vesicles which appear more abundant now in the supranuclear part now in the basal one some existing in this last portion of the protoplasm are in relation to the connective tissue of the crista which is also provided with vacuoles different in size. Each cell has a more or less extended tract of protoplasm connected with the central duct or with one of its branches others the most

superficial delimit the ampullar cavity in which protrude big protoplasmatic digitations whose margins are marked by black granules.

In some points of the reticular zone near the base of the crista can be seen groups of clear polyedric cells sometimes arranged around a central duct (Fig. 1) some of them have a free margin with granules of osmio reducing substance. These cellular groups resemble those of Fig. 2 but have been seen only in a cross section they are surrounded as is characteristic of the reticular zone by cells with a polymorphous protoplasm and a dark small nucleus. These nuclei even 3-4 of them are contained in intercellular spaces of different size and form along with a dark substance that often disguises them.

The cellular organisation found at the base of the crista ampullaris is so clear from the morphological and structural points of view as to permit its being considered as acinous gland.

The importance of this result is remarkable because besides improving the anatomical notion regarding the planum semilunatum it leads to a functional conception different from the classical one since to these glands cannot be attributed anything but a secreting function.

The ampullar fluid seems therefore to have a clear source along with a precise modality of formation one cannot tell however if these glands secrete all the ampullar fluid or as is probable a part of its components.

The doubt about the quality of the product of these glands derives also from the structural polymorphism of the planum semilunatum because since it is constituted of different cellular formations it would be difficult to suppose that all have the same activity. Each of them therefore is supposed to participate in giving origin to the ampullar fluid with a product of its own different from one another as to quality or quantity. Besides it should not be excluded that such fluid may be reabsorbed in a part of the reticular zone. This calls to mind the modality of secretion and of absorption of the cochlear endolymph it does not mean however that identity of chemical composition may exist between cochlear endolymph and ampullar fluid nay I am convinced that the two labyrinthine fluids are different.

The complex structure of the planum semilunatum gives rise to many problems that to day's knowledge does not allow us to solve. It is certain however that nowadays we are interested neither in asserting nor in denying the secreting activity of the planum semilunatum but in ascertaining whether all the zones constituting it are secreting and which is the product of each of them.

It is necessary therefore to investigate further the most important fact is that the planum semilunatum is also furnished with acinous glands.

ZUSAMMENFASSUNG

In jenseitigen Epitheliale Gefilde finden sich im Bereich der Basis der Crista ampullaris die secretorische Drüsen anzusehen sind.

RÉSUMÉ

A la base de la crista ampullaris il y a des formations épithéliales spéciales qu'on a interprété comme glands acinoses

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MALIGNANT CAROTID BODY TUMOUR WITH METASTASES IN THE LUNGS

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In the literature 8 cases of malignant tumours originating from the carotid body with distant metastases have been reported. The author has described one case of a 75 year old woman who died from an acute respiratory infection. The post mortem also revealed a tumour in the left carotid bifurcation and several metastases in the lungs. The histological picture was characteristic of a carotid body tumour. The tumour cells, however, did not show any signs by which a malignant tumour is usually recognized.

Tumours arising from the carotid and the jugular bodies are fairly unusual. These tumours are practically always benign. Certain authors deny that malignant tumours with a capacity to give rise to metastases originate from these organs. Romanowski has gathered the relevant literature in connection with one case of a malignant carotid body tumour. He has found that there have previously been described 7 cases of these tumours with metastases in the regional lymph glands and 8 cases with distant metastases. Among the latter, however, there is one case (Donald & Crile) which does not appear to be satisfactorily documented (1, c. Complete).

These 8 tumours appear chiefly to have given rise to metastases in the liver, lungs and osseous system. The histological picture, both from the primary tumour and the secondaries, was typical of a carotid body tumour. Microscopically the picture was characterized by small cells with relatively small nuclei and sparse acidophilic cytoplasm. The tumour cells gave no positive chromaffin reaction. A very characteristic aspect was that the tumours were rich in the precapillary and capillary vessels which surrounded groups of tumour cells. It is interesting to note that even in the malignant tumours the cells did not reveal any manifest degree of atypia or polymorphism. Mitoses were almost never observed. The tumours were practically always well separated from the surroundings by a capsule.

Since Romanowski's above mentioned work in 1934 no cases of malignant tumours of this kind seem to have been published. In 1938 Rosenwasser described two cases of malignant tumours arising from the jugular body. These would seem to have been the first cases of malignant tumours arising from this organ to be reported in literature. In one of these cases, however, the metastases were only observed by X-ray. There was no post mortem of the patient.

Since malignant tumours arising from the carotid body are a rare occurrence a case will be reported here which would appear to belong to this group

Case Report

Woman born 1880 Suffered periodically from duodenal ulcers since 1932 Treated in 1940 for a duodenal ulcer confirmed by X ray The case history mentions that below the left maxillary angle an *approximately bean sized relatively firm and well demarcated nodule was palpable* The patient was operated on in 1941 at the age of 61 for a chronic duodenal ulcer Blood pressure at this time was 170/100

Operated on in 1946 for double sided benign ovarian fibromata Blood pressure 190/140

After suffering from persistent bronchitis and a heavy cough in 1953 the patient was hospitalized from 21.11.1953 to 13.1.1954 On admission an almond sized nodule was palpable below the left maxillary angle Increased blood pressure 210/110 X ray revealed large densities in the right lung and these diminished considerably after penicillin treatment The electro cardiogram showed left bundle branch block

During 1954 and 1955 the patient had symptoms of congestive heart failure She was re admitted to hospital on 11.1955 Her condition had worsened with increasing dyspnoea The day before re admission there had been a serious deterioration with breathing difficulties On admission numerous rales were heard over both lungs The liver was palpable about 2 inches below the costal margin The previously mentioned nodule below the left maxillary angle was of the same size as before Blood pressure 160/90 After treatment there was a temporary improvement but the patient nevertheless died 19 hours after admission

The *post mortem* (837/55) revealed the following The heart which weighed 290 g was moderately enlarged with manifest left hypertrophy There was moderate arteriosclerosis in the aorta and the coronary arteries There were no changes in the myocardium The valves were unchanged The ostia were of normal width The trachea and the bronchial tree contained a considerable amount of a grey white purulent exudate and the mucous membrane was distinctly reddish in colour

On the left side of the neck fixed in the carotid bifurcation there was a walnut sized well demarcated fairly firm tumour The cut surface was finely lobed and light grey in colour Macroscopically the tumour was clearly separated from the thyroid The lymph glands in the neck were not enlarged soft and without signs of tumour metastases Unfortunately however they were not subjected to histological examination

In the right lung next to the bronchus to the middle lobe and next to a calcified lymph gland there was a tough and distinctly indurated lump about the size of a hazel nut Close to this was another lump about twice the size of a bean consisting of soft tissue with grey red cut surface The bronchus to the middle lobe was considerably constricted by the tumour



Fig. 1. Pulmonary metastases. Tumour tissue to the left. Branch of the pulmonary artery centrally and lung tissue at lower right. H&E, eosin. Magnification $\times 10$.

which, however, did not infiltrate in to the bronchial wall. The middle lobe was completely atelectatic. In all the lobes in both lungs a moderate number of firm granules were visible with a greyish colour on the cut surface. Some of these granules were as large as peppercorns.

In the abdomen changes from previous operations were visible. There were no traces of tumours in the liver, the kidneys, the other abdominal organs or the spinal column.

Histological examination of the neck tumour and the tumour close to the bronchus to the middle lobe showed principally the same picture. The tumour was fairly rich in relatively small, monolayer cells which had small rounded nuclei. The cytoplasm was slightly acidophilic. The tumour cells revealed no manifest degree of atypia or polymorphism. No mitoses were observed. The tumours were rich in small vessels and the reticulum stain revealed groups of cells surrounded by stroma. The tumour cells gave no positive chromaffin reaction when stained by the Savka method (Figs. 1-3).

Here and there in other sections from the lungs there were metastases from the same tumour. Most of the nodules which were visible with the naked eye, however, were lobular pneumonias with an exudate rich in fibrine.

DISCUSSION

The patient who had suffered from hypertension for a long time died as a result of an acute respiratory infection. The histological picture of the metastasized tumour corresponds to that of a carotid body tumour.



FIG. 2 Detail enlargement of a metastatic tumour growth with vessels surrounding groups of tumour cells. V. Gleason. Magnification 150.

It is well known that carotid body tumours grow slowly. In those cases of malignant tumours where the tumour has not been discovered accidentally at the post mortem, the existence of the tumour has been known of for many years before the patient's death. One example of this is the case described by Spolnitz where the tumour was known of for 19 years. It is doubtful how long the patient in the case described here had had her tumour. It is quite possible, however, that it was this tumour that was palpable when the patient was hospitalized in 1940. If this were the case, the tumour had been known of for 13 years.

Authors who have previously discussed malignant carotid body tumours are agreed that neither the primary tumour nor the secondaries show any of the characteristics usually associated with a malignant tumour. In connection with his case of malignant carotid body tumour, Romanski has compared the histological picture with that of a benign tumour. He then observed that in one of the benign tumours there were tumour cells with the largest, most numerous and most bizarre nuclei. Neither in the case described here have the tumour cells revealed any manifest degree of atypia or polymorphism and no mitoses have been observed.

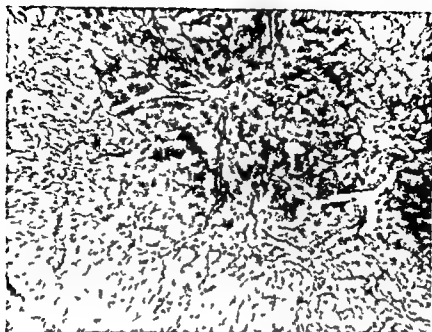


FIG. 3 Reticulum stain of a metastatic tumour growth showing groups of cells surrounded by stroma. Magnification $\times 100$.

This case supports the reports in the literature that there do exist malignant tumours arising from the carotid body. On the other hand such cases would seem to be so rare that it is hardly necessary to reckon with them in terms of everyday treatment. This is further more supported by the fact that histologically these tumours do not show the usual characteristics of a malignant tumour.

ZUSAMMENFASSUNG

Bisher wurden 8 Fälle von malignen Tumoren, welche vom Glomus caroticum ausgehen und in entfernte Organe metastasieren beschrieben. Vom Verfasser wird der Fall einer 75jährigen Frau berichtet, welche im Anschluss an eine akute Bronchitis starb. Bei der Sektion fand man einen Tumor der linken Carotisgabel und zahlreiche Metastasen in den Lungen. Das histologische Bild ist charakteristisch für einen Glomus caroticum Tumor. Die Geschwulst zeigt jedoch im Zellbild keine den malignen Tumoren eigene Struktur.

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O SALA *La cortisone et les composés cortisoniques en oto-rhino laryngologie (recherches expérimentales et cliniques)* Collection monographique 'Minerva Otorinolaringologica', Turin, 1959

This monograph is written in Italian and intended as a thorough survey of the use of cortisone therapy in the ear, nose and throat region. The author points out that cortisone derivatives could be used with advantage on a considerably larger scale than is done at present in this field. To support his opinion the author quotes a number of experimental examinations, particularly of the formation of new connective tissue, of induced corrosive damage to the oesophagus in rabbits, and of the effect of cortisone treatment in various allergic conditions. These experiments form the basis for clinically developed methods of treatment with cortisone derivatives in a number of different pathologic states within the field of otology.

The book is extremely interesting even if the language prevents its being widely read. Many of the experiments are well planned and carried out. The conclusions drawn are, however, in many cases rather far fetched. The clinical material is homogeneous in all respects, if somewhat small. The author is very optimistic about the use of cortisone derivatives. If the doses recommended by him are used certain risks are inevitable and it is questionable whether these are justifiable in, for instance, cases of acute otitis, intubation granuloma, or in certain so called laryngopathies. In slight cases of suspected corrosive damage to the oesophagus too, cortisone treatment must be considered of doubtful value. We have learnt from Swedish endocrinologists to be more restrictive, even in allergic conditions.

The use of cortisone in plastic operations of the tympanum is interesting, but the effect there is bound to be still more doubtful. Interesting too are the author's views on the effect in cases of stapes mobilization. There is certainly much further research to be done on this subject.

The criticisms do not lessen in any way the value of the book. It deserves great attention both as a reference book and for the ideas contained in it.

H. Diamant

JOSEPH WATZKE *Ein binauraler Horsynthese Test zum Nachweis cerebraler Hörstörungen* Georg Thieme Verlag Stuttgart, 1958. 117 pages, 46 ill. Dfl 19.80

This paper describes a method for proving cerebral auditory defects.

The first part begins with a short description of the anatomy of the ear and the auditory paths in the brain and, after a survey of what is hitherto known about

a valuable survey of the methods already available for indicating cerebral auditory defects.

In the second part of the paper the author describes his method of dividing speech into two frequency areas—as did Fletcher (1929)—and leading one area to the right ear and the other to the left. After several experiments a method was evolved using a sound tape containing 41 words, preferably of two syllables, of which one ear

receives frequencies between 500 and 800 Hz and the other ear frequencies between 1500 and 2100 Hz. If the patient hears more than 10 per cent of the words wrong the aural synthesis is considered abnormal. In a second test both the frequency areas are led to both ears at once, using the same 11 words, and the errors are counted. A third test is then made, identical with the first test. Finally the three sets of errors are assessed according to certain specified norms.

The third part of the paper contains a report of experiments with 750 patients involving whispering and normal voice tests, binaural tests as described above, and—in most cases—tone audiograms. In persons over 65 years of age the binaural test was usually positive, i.e. pathologic. In children the test was often positive without demonstrable injury or disease. Differences in intelligence did not appear to affect the binaural test. Out of 18 patients with verified tumours, abscesses, cysts, metastases or aneurysms of the brain the binaural test was pathologic in 12 cases, two were described as borderline cases, while only four cases showed negative results. Eleven out of 16 patients with atrophy of the brain showed positive binaural tests. Out of 36 patients with chronic hypertension, 21 showed positive tests and four were borderline cases. Five out of 11 patients with multiple sclerosis showed positive test results, while four were borderline cases. Similar examinations are also reported of patients suffering from head injuries, epilepsy, psychoses, cerebral circulatory disturbances, migraine, encephalitis, intoxications and metabolic disturbances. Finally reference is made to the damage to the brain stem discovered by Huchcs in cases of disease and injuries in the brain. The author believes that this explains why, for instance, injuries to the cortex also cause disturbances in the aural synthesis.

Examinations have been made—by this binaural method but using Swedish words—at the Otological Department in Gothenburg and the results have been published but these have not been able to prove the value of the method.

U. Kuhler

C. I. PORTA *Manifestazioni Emorragiche in Otorinolaringologia* Scuola Ippocratica Benedettina, Parma, 1960. 161 pages.

This book, published by the Italian Society of Otorhinolaryngology, under the direction of Professor Carlo Felice Porta of the University of Parma, consists of a fairly elementary survey of spontaneous, accidental (postoperative) haemorrhages from various ear, nose and throat sections. Aetiology, pathogenesis and treatment are gone into in detail and the haemorrhagic complications analysed in such operations as the otologist may be likely to carry out, together with the measures necessary when treating these conditions. The book is illustrated with numerous plates and colour photographs. A comprehensive list of references is contained in a 21 page appendix. As it is written in Italian the book is likely to be inaccessible to many readers.

E. H. Brunell

SCHUCHARDT K. *Fortschritte der Kiefer- und Gesichtschirurgie* 1 in Jahrbuch, Band VI Georg Thieme Verlag, Stuttgart.

This volume deals with Pathology, Diagnosis and Therapy in Diseases of the Mandibular Joints and the Salivary Glands. The individual contributions are papers read before the Congress of the German Association of Mandibular and Facial Surgeons in Düsseldorf in 1959.

After an introductory address surveying the anatomy and physiology of the mandibular joints and the roentgen diagnosis of their diseases, Husted discusses the importance of arthrography when diagnosing disorders of the mandibular joint. He considers that this examination, introduced by Nørgaard, is technically difficult and that the interpretation of the roentgenograms demands great experience. The method is therefore only practicable in the specialised departments of large hospitals, where sufficient experience may be gained.

Displacement, arthrosis and ankylosis of the mandibular joints are also discussed, as are various operative methods of reduction. Schuchardt stresses that good results may usually be obtained with conservative, orthodontic treatment, thereby avoiding operation. In arthrosis of the mandibular joint the intra articular injection of hydrocortisone, hyaluronidase, trypaflavin and rivanol, and other agents have been given a trial usually with favourable results. Removal of the articular disc, on the other hand is only considered advisable in occasional cases which are not amenable to conservative therapy. The operative treatment of mandibular ankylosis is discussed in a number of papers and different methods of interposition are described. Schröder, from Schuchardt's department, considers interposition unnecessary and stresses the importance of subsequent functional therapy for at least three to four years.

The last part of the book contains over twenty papers concerning the salivary glands and their diseases. Redon reports on the treatment of carcinoma of the parotid gland. Of nearly 800 parotid tumours, some 20% were found to be carcinomata. As the diagnosis can only be made histologically, he emphasizes the importance of every parotid tumour, however small, being operated upon as early as possible.

The general impression of this new volume of *Fortschritte* is very good. The typography is admirable and the illustrations well chosen and mostly of high quality.

B. Islam

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